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THE RESULTS OF 76,000 ADENOID AND TONSIL OPERATIONS.*

DR. WILLIAM H. TURNLEY, New York.

This is a big subject to cover in 15 minutes; however, "brevity is the soul of wit," so I will try to be brief and state only the results, without any discussion, of this series of operations. My conclusions are not being influenced by any report of previous operations here or elsewhere. They were all done in the Tonsil Clinic which Dr. David Jones so thoroughly organized at the Manhattan Eye, Ear and Throat Hospital here in New York City. They extend over a period of seven years and are all ambulatory cases. The youngest patient was 6 months of age; the oldest 72 years. They represent every race, color and creed, from 61 different countries.

Why is there so much discussion regarding tonsils? Chiefly because we do not know all about their function; but we do know something about results after removing them. In this series I have not seen a single patient who has suffered any ill effects from having his tonsils removed; that is, from a general standpoint of disturbed function. There are a few minor discomforts occasionally, such as a dry throat or a sour taste resulting from scar tissue formation. A few patients have a slight speech defect for a short time, due to paralysis of the soft palate. Some may have more bronchial colds after

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their tonsils have been removed, instead of head colds, as previously.

	1931	1932	1933	1934	1935	1936	Total
Patients examined	14,321	14,301	12,779	12,994	12,770	10,584	77,749
Patients operated	11,925	12,001	10,583	10,698	10,212	9,834	65,253*
Postops. examined	10,075	10,151	8,834	9,522	8,937	9,464	56,983
Rheumatic history	721	776	694	636	1,010	982	4,819
Rheumatic pts. op.	613	668	586	528	592	559	3,546
Rheumatic postop.	553	604	516	469	518	512	3,172*
Per cent rheum. pts.							6%

The table shown here was arranged primarily from the standpoint of rheumatoid cases. The term "rheumatism" in its broad sense is meant to include painful conditions of the muscles, fascia, bones, nerves and joints. In this series are included 3,172 patients who have been followed for the past six years; and of these 83 per cent are improved or cured; 12 per cent show no improvement and 5 per cent are worse. Of those that showed no improvement or became worse, all had some other foci of infection, such as carious teeth and sinusitis. Also they were past their youth or had some bony changes taking place. The younger the patients and the shorter the duration of the rheumatism, the better the results, and vice versa. The largest group consists of those between 30 and 45 years of age.

Patients suffering from heart lesions have not been followed as closely; but I do not believe that they are benefited as much as the rheumatic patients. The general opinion is that patients with heart lesions do not tolerate ether so well, but in 1,000 observed cases they do not seem to have shown any ill effects. We have not lost a single heart case in this series. I hasten to add that most of the patients had purely valvular lesions. No decompensated heart cases were operated upon.

The removal of tonsils lessens the incidence of sore throat about 90 per cent, for the reason that a sore throat is fundamentally tonsillitis. No cases are operated upon within 10 days following an acute attack. Keratosis leptothrix is usually cleared up by the removal of tonsils; likewise a stubborn Vincent's ulcer of the tonsil. Nonspecific chronic laryngitis is greatly helped. Naturally, a quinsy cannot develop after a tonsillectomy unless there is a piece of tonsil tissue left; but an occasional retropharyngeal abscess may occur.

As for the ear, the incidence of otitis media in children is reduced 95 per cent by a thorough adenoidectomy. In adults, tubal catarrh is greatly improved by the removal of adenoids; but otalgia is less often relieved.

Certain patients with involvement of the uveal tract of the eye, such as iritis, are benefited. Chronic conjunctivitis is quite often cured, while optic neuritis is less often helped.

Sinus infections are benefited in a general way. In fact, all pansinusitis cases have a preliminary tonsillectomy performed. If not, they are almost sure to develop an acute tonsillitis following the sinus operation. And please remember that sometimes the postnasal discharge in adults may be the secretion from an infected piece of adenoid tissue which has never been removed, and may not be from a sinusitis at all.

Children who have a general systemic rundown condition, as evidenced by backwardness in school, are tremendously improved by an adenoid and tonsil operation. Almost immediately they show an improvement in their studies, as well as in their color, weight and appetite. Children's diseases, as diphtheria and whooping cough, run a much milder course if the tonsils and adenoids have been removed previously.

Patients with involvement of the thyroid gland are benefited, as shown by the failure to respond to a thyroidectomy until their diseased tonsils were removed. Tuberculosis of the lung has shown complete calcification in a few months following an adenoid and tonsil operation. I have never seen agranulocytic angina in a patient who had no tonsils. Whether tonsils have anything to do with the cause, I would not like to say. The true allergic cases, such as rhinorrhea, rose cold and hay fever, are not responsive to any appreciable extent; but children with asthma may be greatly benefited. Adults with asthma are occasionally helped.

Status lymphaticus is almost negligible; and I sometimes wonder if there is such a condition. An occasional enlarged thymus is encountered on observing the patient in the clinic; while a few others are shown on a lateral and front view X-ray film; however, the patients seem to stand the operation very well. We try to avoid operating on women within five days of their menstrual period and give them written instruc-

tions to that effect; but from observing those that have slipped by, I have not noticed that it made any difference. The coagulation time does not seem to show any definite indication as to the possibility of hemorrhage. In fact, there are very few contraindications to a tonsillectomy; however, all adults over 50 years of age are given a thorough physical examination, and we do not operate on children under 2 years of age, except where we have obtained a history of repeated colds and sore throat with enlarged glands of the neck, otitis media, etc. In such patients the adenoids are removed, and occasionally the tonsils, at the discretion of the surgeon in charge.

Hemorrhages are the most important complications; but they are all checked by pressure, ligature or an occasional suture. Some are a little more difficult to check than others. Adult patients, and those who have been operated before, either by surgery or electrocoagulation, usually cause more trouble than children and primary tonsillectomies. There are more bleeding vessels to ligate at the time of operation during the summer months, when the barometric pressure is low. A child may bleed during sleep and become exsanguinated very quickly, necessitating a transfusion. We believe that blood transfusion, repeated if necessary, is the ideal treatment for hemorrhage. Fresh hemorrhages are usually due either to ligatures slipping off when the patient gags and coughs, or to the inability to see and tie a vessel at the base of the tonsil during operation. Hemorrhage after a few days is due to infection. Most of the drugs used to prevent or check hemorrhage are of little value, so far as can be determined. Snake venom has not been tried in our hospital. We have not lost a single patient from hemorrhage, nor have we had to ligate the carotid artery.

Tonsils do not grow back any more than any other gland. Pieces are sometimes left, especially when the Sluder instrument is used to obtain speed, or when there is not complete anesthesia, or when the light is poor. A piece of tonsil left at the upper pole is more likely to be caught in scar tissue and form an abscess later; while a piece left at the base shows more of a tendency to hypertrophy. Sometimes a tonsil remnant is more lethal than the entire gland. When a fossa becomes infected, then granulation tissue may pile up, resembling almost a whole tonsil; but there seems to be very little, if

any, absorption from this type of tissue should it subsequently become infected. Quite often, if adenoids are removed in children too young, there is a compensatory hypertrophy of lymphoid tissue extending all over the naso- and oropharynx.

More incomplete adenoidectomies are done than tonsillectomies; chiefly, I believe, because we cannot see so well, or do not bother to feel up in the nasopharynx afterward. Also, some fingers are not sensitive enough. Hemorrhages from the adenoids are usually stopped by sponge pressure, or by removing an organized clot or a piece of adenoid tissue.

Postoperative patients are kept in the hospital over night; the following morning they are allowed to go home if they do not have a temperature above 100.4° or if they have not had a hemorrhage, unless for some other reason we do not consider them well enough to go. They are each given a slip of directions as to their care at home, such as rest, diet, laxative, etc. They are requested to return at the end of a week for inspection and again after three months. Children are given a 10 per cent solution of argyrol, of which two drops are put in each side of the nose, twice daily, for three days. Adults are given a warm Dobell's solution to use as a gargle. Aspirin chewing gum is prescribed for all. It helps prevent the muscles becoming stiff and relieves the pain to some extent. About 50 per cent of the patients have a reflex pain to the ear for a few days, and about one-half of 1 per cent develop an acute otitis media as a result of infection in the adenoid region, following an adenoid and tonsil operation.

The number of lung abscesses encountered after an adenoid and tonsil operation is remarkably small, about four a year, or one in 3,000 cases. We have had only one foreign body inhaled into the lungs as a result of these operations. That was a bicuspid tooth which was dislodged by the mouth gag. It was coughed up later without any serious result to the patient. Following this accident, all loose teeth are removed before operation; but should one come out, or a piece of instrument be broken off, then an X-ray of the chest is made immediately.

The last 15,000 cases have been given a mixture of CO_2, 10 per cent, and O, 90 per cent, to inhale before leaving the operating table. This not only seems to revive them more

quickly, but the incidence of cough irritation is lessened. It is spoken of as de-etherization.

Our deaths total six for the 76,000 cases, or about one in 12,600. None of them was from hemorrhage, and there have been no deaths in the last two years from any cause. One of the six was a hemophiliac, but died of pneumonia; one died of heat prostration; one from acute edema of the thymus; one from septicemia; one from ether narcosis; and one was reported as status lymphaticus.

In general, our results have been excellent, and I advocate the removal of tonsils in all rheumatic cases. I think also that if children had their tonsils and adenoids removed before 5 or 6 years of age, there would be less chronic sinusitis in adults, and we would raise a much healthier race of individuals here in the temperate zone.

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CHOICE OF OPERATION IN TONSILLECTOMY.*

DR. JOSEPH D. KELLY, New York.

Those of us who have had sufficient experience with tonsil operations know that it is very difficult to apply any definite operative procedure to all types of tonsils. It may be said that the tonsil operation can be the simplest and easiest of surgical procedures in some cases, and in other cases will require all the skill and judgment we have at our command. The very fact that we have such a variety of mechanical devices for the removal of tonsils is sufficient evidence that laryngologists over a long period of time have been trying to find an easy and fool-proof operation. The basis of nearly all of these ideas has been the Matthew's tonsillectome or some combination of this with the snare. The Sluder instrument has had many modifications and one of the latest inventions is a combination of the Sluder and snare. In late years with the publication of the carefully compiled records of some of the large tonsil clinics, we have been made cognizant of the fact that many of our apparently slick looking Sluder operations were not so slick looking six months or a year after. Some men have reported as high as 73 per cent incomplete operations. Hence, within my knowledge during the past 10 years, there has been a gradual swing of the pendulum away from the machine operation to a more surgical and careful dissection.

Many of us have experienced the difficulty of meeting anomalous vessels in the tonsillar fossa, both venous and arterial, which have required the most extended technique and judgment to finish our operations completely without jeopardizing the life and comfort of our patients. It is, therefore, fitting and proper that we should have techniques to fit the particular case at hand and to be able to apply any modification thereof or any extra procedure that exigencies of the moment may require.

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For the sake of clarity and for practical purposes, I believe that we may classify the tonsil into two large groups: First, those of children, and secondly, those of adults. We further subdivide each of these into groups one, two and three, depending upon the anatomical characteristics of the tonsils.

CHILDREN'S TONSILS.

Under group No. 1, we refer to girls and boys under the age of puberty and we speak of that type of tonsil which may or may not be large, but which is cryptic, filled with retained secretions, and is completely, or almost completely, submerged between the tonsillar pillars. In addition to being submerged, sometimes this particular type is quite adherent to the tonsillar fossa because of repeated attacks of tonsillitis, and is difficult of displacement.

Under group No. 2 of children's tonsils might be defined those tonsils which are of moderate size, cryptic, filled with retained secretion, and moderately hidden by the tonsillar pillars, but which, on manipulation, are easily dislocated from the tonsillar fossa.

Under group No. 3 may be designated that type of enlarged, hypertrophied, pendulous, cryptic tonsils which set well out from the tonsillar fossae and are only partially covered or hidden by the tonsillar pillars.

In making this classification we endeavor to form some criterion by which to judge the operative procedure best suited to a particular case.

The operative procedure in class one is, without question, that of a careful dissection with or without the use of a snare. The method of dissection will vary with the inclination of the operator and the effectiveness of his particular technique. There is no accepted method of dissecting which is applicable in every case and, hence, the operator proceeds with that method of dissection which has given him the best result. Where some men will use scissors, others may use some form of serrated dissecting knife or other type of instrument. In the act of dissection the individuality and the dexterity of the operator determines the speed of his dissection and the amount of trauma to the tissues.

In the Jones' Clinic at the Manhattan Eye and Ear Hospital, we have been taught that the most essential step in the procedure of dissection is to define as quickly as possible and with the least trauma the capsule of the tonsil. Whether this definition be started at the upper or lower pole of the tonsil is dependent upon our ability to dislocate the tonsil from its fossa. If it can be dislocated sufficiently to stretch the plica semilunaris to a point where there is a good delineation of the upper pole of the tonsil, then dissection is begun at that point. If this cannot be done, then we have found from experi-

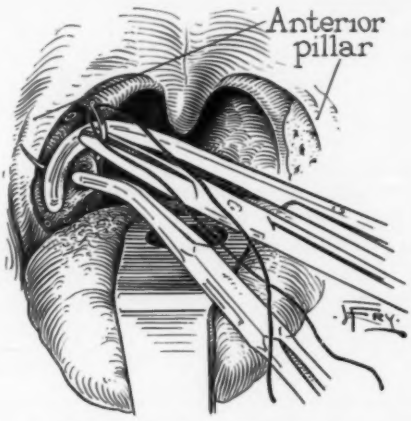

Fig. 1. Method of suture ligature.

ence that the next best procedure is to elevate the anterior pillar by the use of a Matthew's knife, a Silar's knife or other instrument which will perform this function with a minimum of trauma until we are able to distinguish the upper pole of the tonsil. From this point the posterior pillar is elevated and the capsule of the tonsil separated carefully from the tonsillar fossa by the use of tonsil scissors or separator which will carefully dissect off the palatopharyngeus muscle from the posterior surface of the tonsil. Under certain conditions this may be done rapidly and safely by the use of the Fisher knife or some modification thereof.

I believe that this operative procedure will suffice to handle most every case of tonsillectomy required in group No. 1.

In group No. 2, we will meet the borderline cases which overlap from group No. 1 and it is always best to apply the surgical procedure for group No. 1 to the doubtful cases in group No. 2. Where the cases in group No. 2 are clinic cases and where the object of the procedure is to give the greatest good to the greatest number with the least possible time, then I should say that the use of the Sluder instrument or any modification thereof would be indicated. If the case is a private one, and the patient is paying well for your time and effort, then I believe all cases in group No. 2 will have a better result and an easier convalescence if the Sluder is not used, and the operative procedure of group No. 1 is applied. In group No. 3, where we are dealing with a large, pendulous, compact mass of lymphoid tissue with a small attachment to the fossa, very little adherence of the tonsil pillars, and where there is no danger of injuring the muscularis and little chance of leaving a piece of tonsil, I should say that the Sluder or the Sluder-Beck operation may be safely performed. In most of these cases there is usually a large amount of lymphoid tissue at the base of the tonsillar fossa and it is very necessary that this tissue be removed. There was a time when some outstanding laryngologists recommended leaving this tissue to help convalescence and relieve pain; however, the other members of our profession became so loud in criticism that we are obliged to remove it in self defense.

The amount of hemorrhage following any operative procedure depends in many cases upon the individual potentialities of the patient and the amount of trauma at the time of operation. There are a number of methods used for the control of hemorrhage in children. To my mind, the safest and most expedient method of handling hemorrhage in children is at the time of operation. If there be any question as to whether or not the patient may bleed in the near future, it is wise and prudent for us to stop all bleeding points by the use of a suture ligature before he leaves the operating table. Children are entitled to as much care and consideration from a hemorrhage standpoint as adults.

ADULT TONSILS.

The type of tonsils in the adult does not differ much from those we find in the children, except perhaps that in the adult we see a less number of tonsils which might be classed under

group No. 3 of children. The group No. 3 of the adult tonsil, while not as pendulous as that of group No. 3 in the child, nevertheless, for its size, is, relatively speaking, just as large, if not larger.

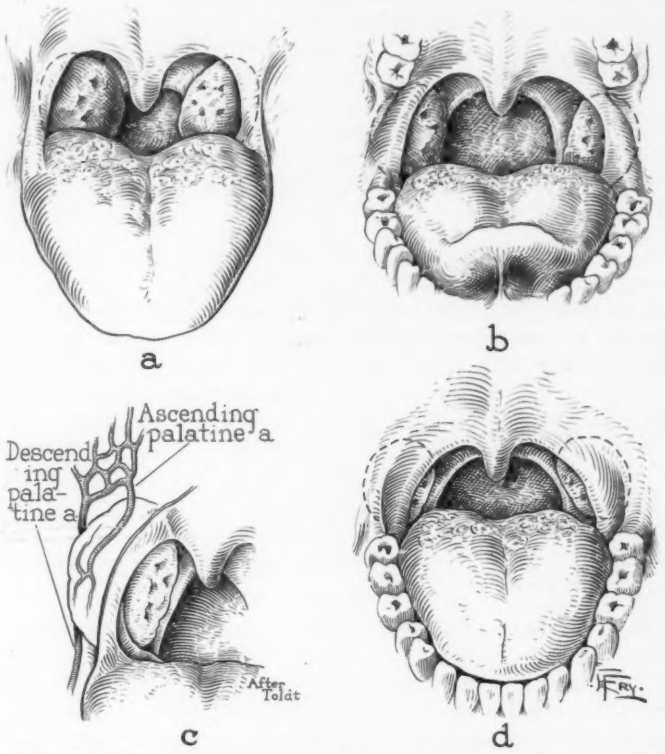

Fig. 2. Type of tonsils. (D) Group No. 1. (B) Group No. 2. (A) Group No. 3.

a. In group No 1. of the adult we have to deal with the buried, submerged, or covered tonsils. These are usually found in individuals who say they have had several doctors examine their throats and have been told that they have no tonsils. In these cases we see a condition in which the tonsil pillars practically hide the whole substance of the tonsil and it is necessary to retract the anterior pillars or dislocate the tonsil

from its bed in order to determine its size. If, in addition to the tonsils being buried, the patient has suffered from numerous attacks of tonsillitis, or if the tonsils are in a state of chronic inflammation and are very adherent to the tonsillar fossa, we are confronted with one of the most difficult types for tonsillectomy. If we have made an effort to determine beforehand and are convinced that we are dealing with such a case, I would say for the safety of the patient and the anxiety of the operator that this case should be handled under general anesthesia and by careful dissection. While it is possible to perform this operation satisfactorily under local anesthesia, nevertheless, there may be undue shock to the patient and some very anxious and embarrassing moments for the operator.

b. Under group No. 2 in the adult tonsils, may be placed that type of tonsil of moderate size which is only partly submerged. This type of tonsil presents about one-third of its size for examination while two-thirds of it is covered by the tonsillar pillars. It may or may not be adherent, but should be sufficiently movable that we may attain a good delineation of its borders, so that a definite line of cleavage may be expected at the time of operation. This type of case is best handled by the dissection method; however, a fair percentage may be handled satisfactorily by some one of the other methods. If the tonsil is not readily dislocated and complete relaxation is necessary, then I believe a general anesthetic should be given.

Whether these cases should be handled under general or local anesthesia depends, I believe, very much on the skill of the operator and the inclination of the patient. If the operator is quite dexterous and the patient is a composed, stable individual and does not want general anesthesia, then I see no reason why a very large percentage of these cases cannot be handled under a local anesthetic.

c. Under group No. 3 we can place those adult tonsils which are usually large, hypertrophied, cryptic and which, in addition to being diseased, may offer mechanical obstruction, etc. This type of tonsil is very often easily dislocated forward or backward in the tonsillar fossa. One might be of the opinion that it would be safe to use some type of guillotine instrument for its removal; however, we have learned from experi-

ence that these cases are best handled, whether under general or local anesthesia, by the use of dissection and snare. While it is possible to use a form of guillotine instrument in some of these cases, it is better not to; for there is a very good chance of leaving a piece of tonsil at the upper or lower pole or posterior surface of the tonsil which may be overlooked at the time of operation and not become evident until some time later. Many of these cases are best handled under general anesthesia because the tonsils are so large that they are sure to have a generous blood supply. It will undoubtedly be necessary to use two or more suture ligatures to control bleeding, and we are always more comfortable stopping a hemorrhage while the patient is under general anesthesia.

In this classification nothing has been said about the operative procedures for secondary tonsillectomy. To my mind, there is only one procedure to be used in this type of case, and that is a careful dissection and snare or careful dissection. In very few cases is it possible to use any form of guillotine instrument because of the scar tissue present.

In making this simple classification on the types of tonsils to be dealt with surgically, our object is not to criticize any other classification that may seem more logical and acceptable, but to outline to you the method that we use in anticipating our sugical procedure in a given case. Our surgical procedures are those which we have found most efficient and effective in the handling of our own problems and we present them for your consideration and criticism. Procedures and mechanics of tonsillectomy will differ in a thousand ways and we all hope to learn from the experience, methods and failures of others.

140 East 54th Street.

ARGYROSIS.*

DR. CARL KAPLAN, Brooklyn.

This may be defined as a form of chronic silver poisoning. It is not serious. This may be brought about by administering silver or its protein compounds by mouth or by application in the form of nasal drops, or eye drops. Argyria is quite incurable. Many attempts have been made to cure it but without success; iodides have been tried but without success. The only known solvent of silver granules is cyanide of potassium. Since this drug is so powerful and poisonous it is absolutely impossible to employ it. Potassium iodide may be tried in curing cases of argyria.

Years ago the chief source of chronic silver poisoning or argyria was found in the treatment of epilepsy. In those days silver was used as a therapeutic agent to cure this ailment. Of late, cases have been reported in individuals who make artificial pearls. This industry uses silver as a pigment.

In my cases the source has been from the continued use of argyrol and neosilvol. It was employed in combination with other ingredients as nose drops. Each case being reported tonight used these preparations constantly for more than two years.

The deposit of the silver granules into the skin or mucous membrane gives the color varying from light gray to dark, bluish-slate color. It is distributed all over the body, especially the face. It is mostly present in the corium rather than the epidermis. The pigmentation is present also in the alimentary canal and respiratory organs, the glomerule of the kidneys, the connective tissues of the liver and spleen, the choroid plexus and the tunica intima of the aorta. The pigmentation does not produce any other symptoms. The patient is perfectly comfortable and may live to a ripe old age.

Cases of local or general argyrosis are very rare and this fact prompted the author to present these two interesting

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cases. The literature is very meager in reports of similar cases. Only two cases have been reported. One case is described as involving the nasal mucosa alone. It is more frequent as argyria of the conjunctiva.

In the two cases of argyrosis of the nasal mucous membrane one followed the application of 5 to 20 per cent silver protein solution daily for eight or nine years. This produced a discoloration of the membrane of the nose, having a distinct bluish-gray appearance. The second case followed the application of mild silver protein solution in the nose without medical advice. In these cases the skin, mucous membrane, conjunctiva and sclera were involved.

Case 1: L. F., age 44 years, female, housewife. *Chief Complaint:* Nasal obstruction, postnasal drip. *Examination of Nose:* Atrophic rhinitis. The mucosa of the nasal mucous membrane on first examination was pale, covered with many crusts, mucopurulent secretion in both middle meatus. *Medication:* Adrenalin solution 1/1000, dr. 1, novocain 4 per cent, dr. 1, neosilvol 20 per cent, q.s., oz. 1. Patient continued to use these drops from 1926 to 1929. At that time the examination of the nose showed that the mucous membrane was stained a bluish-slate color. This was especially marked in the mucosa of both inferior turbinates. The skin over the face, conjunctiva and sclera, as well as the rest of the body, also assumed a bluish tint.

A diagnosis of argyrosis was made; the patient was instructed to stop using nasal drops. I kept this patient under observation for the past six years and up to the time of writing this paper the discoloration has not faded.

Case 2: G. R., age 8 years, male. Patient was admitted to the Jewish Hospital, Pediatric Clinic, June, 1932. *Chief Complaints:* Cough, asthmatic attacks and frequent head colds. Argyrol 15 per cent was used from April, 1932, to October, 1934, at different periods. On Oct. 8, 1934, examination of the child's nose showed that the mucosa over both inferior turbinates, especially the right, were stained bluish in color and remained so ever since. The mother was instructed to stop using the argyrol drops. Up to the present time the argyrosis is still present.

Conclusion: One must be cautious in using any silver medication, or silver compounds. The patient must be instructed not to use these argyrol and neosilvol drops continuously. Many patients have the habit of renewing their medication without consulting their family physician. They must be warned against self medication.

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CORRECTION.

Dr. Albert A. Cinelli, of New York City, asks that the following correction be made in his article, "Labyrinth Reactions and Their Clinical Importance," published in THE LARYNGOSCOPE for November, 1936:

Fig. 11 shows the endolymphatic movement away from the ampullated ends; therefore, the slow or vestibular component is toward the *opposite side,* whereas Fig. 12 shows the endolymphatic movement towards the ampulla, with the slow or vestibular component towards the *same side.*

This paragraph should read: Fig. 11 shows the endolymphatic movement away from the ampullated ends; therefore, the slow or vestibular component is toward the *same side,* whereas Fig. 12 shows the endolymphatic movement towards the ampulla, with the slow or vestibular component towards the *opposite side.*

IS IONIZATION WORTH WHILE FOR THE ALLERGIC PATIENT?*

DR. ARTHUR M. ALDEN, St. Louis.

My experience with ionization began in September, 1933, when, following the technique elaborated by Warwick, I treated 19 patients with hay fever, all of them in the midst of their symptoms. The relief in each case was prompt and complete for the remainder of the season. These two factors seemed at that time to warrant a pardonable optimism as to the prospective value of this method in the treatment of the nasal manifestations of hay fever and perhaps of the other forms of allergy. Accordingly, we began a series of cases to determine, if possible, just what the therapeutic limitations of nasal ionization were.

Now three years have elapsed and a sizable literature upon this subject has appeared in the current medical journals. As the direct result of the Frank E. Ball Research Fund, many valuable contributions, not only to the better understanding of ionization, but toward a more adequate comprehension of the role of allergy in rhinologic practice have been made possible. This investigation has been carried out and published by the personnel of the Oscar Johnson Institute at Washington University School of Medicine and constitutes one of the most important recent contributions to the rhinologic aspects of allergy. In addition to this many other publications have appeared, dealing with the clinical phases of ionization. In the light of all this accumulated information can we now evaluate this procedure with a reasonable amount of accuracy? I think so.

Errors were made in the beginning which have been corrected as more information accumulated. At first, all of the work was done following the complicated technique advocated by Warwick. This included the motor generator machine and zinc-tin-cadmium electrolyte solution. It was soon found that

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any source of smooth galvanic current was suitable and that 2 per cent zinc sulphate solution gave the same results as the trimetal electrolyte. When the zinc sulphate was incorporated into a tragacynth jelly and instilled into the nose with a syringe and cannula, it greatly reduced the trauma which followed the original method of nasal packing and lessened the discomfort and reaction of the patient to the treatment. All of our later work has been done in this way.

There is now no mystery as to just what ionization does. Its application to the nasal mucosa produces a mild coagulative necrosis of the superficial layer of the mucosa with polymorphonuclear infiltration of the submucosa and a residual deposition of connective tissue fibres in the tunica propria. This results in squeezing out some of the glandular elements and thus lessens nasal secretion. There is still some disagreement among authors as to the final effect upon the ciliated layer, but I believe that it is safe to say that it returns to about what it was before the ionization. None of the noses that require treatment are normal and many of them show extensive replacement of the ciliated cells by those of lower cuboidal and even squamous architecture before any treatment is given. The end-result clinically is a nasal mucosa that is somewhat dryer than formerly and much less subject to intermittent attacks of intumescence.

We know that ionization in no way alters the fundamental physiological characteristics of the individual that go to make him allergic and for only a relatively short time does it even decrease the ability of the nasal cells to combine with and be affected by their offending allergens. What it does do is by a mechanical change to render the nasal mucosa less able to produce disagreeable obstructive and secretory symptoms in response to either external irritation or vasomotor stimuli.

With this last statement clearly in mind it is a simple matter to give ionization its proper place as an adjunct to rhinologic therapy.

Now as to the evalution of the clinical results. My own estimation of the relief following ionization in hay fever and other allergic conditions, published in the spring of 1935 and based upon the results obtained during the season of 1934 and half of the 1933 season, was all wrong because not enough time had elapsed for the patients to resensitize themselves. At

the present time resensitization has taken place in most of my hay fever patients and they still have their symptoms at the appointed time, though a number of them, due no doubt to the fibrotic changes in their submucosa, say that their nasal discomfort is much less severe than formerly.

Certainly, desensitization is still the method of choice in the treatment of this condition, with ionization being reserved for those cases where the obstructive and secretory symptoms are predominant and where the mechanical relief to be expected is more than commensurate with the tissue damage incident to the ionization.

Asthma has not been lessened after ionization except in a few cases, where I am sure that the improvement was due to the relief from nasal obstruction rather than to any change which the treatment brought about in the allergic status of the patient.

The symptoms of perennial allergic rhinitis have in many cases apparently been greatly lessened following ionization.

When we stop to consider that the nasal mucosa of the adult patient with this condition, which in most cases began early in life, represents merely the summation of the tissue responses to innumerable allergic insults, we are not surprised when a biopsy specimen of it reveals what appears to be almost irreparable damage. The surface epithelium has lost much of its cilia and many of the superficial cells have undergone metaplasia in the direction of their replacement by cells of lower cuboidal or squamous type of architecture. The glandular elements are hyperplastic and frequently cystic. The walls of the blood vessels have lost their elasticity and the bulk of the soft tissue in the nose has so increased that comfortable nasal respiration is impossible, even during the periods of remission of allergic activity.

It would seem that any procedure that would alter this picture in the direction of lessening the bulk of the edematous mucosa, reducing the glandular hyperplasia and giving the patient a roomier, dryer nose would be acceptable. The deposition of a little fibrous tissue in the submucosa such as follows ionization is just about enough to squeeze out the cystic contents of the glands, reduce the edema, give the needed support to the flabby walls of the vascular apparatus and make

the patient comfortable. I know of no method of treatment that accomplishes this result better than does ionization.

To summarize:

1. Ionization produces in the nasal mucosa a mechanical change which renders it less able to produce profound vascular or secretory responses to either external irritation or vasomotor stimuli.

2. Ionization does not alter either the allergic status of the individual or the ability of the nasal cells to be affected by allergens.

3. The value of ionization as a therapeutic method in rhinology rests solely upon its efficiency as an agent to reduce excessive nasal intumescence and hypersecretion.

537 Frisco Building.

PHARYNGO-MAXILLARY FOSSA; METHODS OF OPENING THROUGH THE MOUTH. CASE REPORTS.*

DR. HENRY M. GOODYEAR, Cincinnati.

Very little stress is laid upon the importance of the pharyngomaxillary fossa in the general teaching of anatomy; in fact, you may have difficulty in finding mention of it in a number of excellent texts on anatomy. Mosher[1] states that it is more of a potential than an actual space until it is filled with pus.

BOUNDARIES.

1. It is a cone-shaped space with the base attached to the skull about the jugular foramen. *2.* The apex is at a level of the hyoid bone extending as an envelope (carotid sheath) along the great vessels into the thorax. *3.* Laterally is the internal pterygoid muscle and a small portion of the parotid gland. *4.* Medially lies the superior constrictor muscle with the tonsil attached to it. *5.* The fossa is bound posteriorly by the prevertebral muscles and the prevertebral fascia. This *fossa,* however, is divided by the pedicle of the neck viscera into the pharyngomaxillary *space* anteriorly and the retropharyngeal *space* posteriorly by this pedicle. *6.* Anteriorly the space is limited by the pharyngomandibular raphe.

The pedicle of the neck viscera includes the following important structures; namely, *a.* Internal jugular vein; *b.* carotid artery; *c.* styloid muscles; *d.* cervical sympathetic chain.

This pedicle divides the fossa into unequal parts, the anterior, the true pharyngomaxillary space, and the posterior retropharyngeal space, the latter containing the lymphatic glands involved in retropharyngeal abscesses in children.

The mass composing the visceral pedicle makes it impossible to pass from the pharyngomaxillary space into the retropharyngeal space without the use of force (Oscar V. Batson, Philadelphia).

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*Read before the Cincinnati Otolaryngological Society, May 21, 1936.
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INFECTION.

August L. Beck found that the pharyngomoxillary fossa was infected directly, or became infected, in over half of the cases of severe neck infections.

It may be infected by an extension from the pharynx or tonsils, retropharyngeal abscess, parotid or submaxillary spaces, cervical vertebra, the mastoid tip and petrous bone.

SYMPTOMS OF INFECTION.

1. Trismus occurs early due to splinting of the internal pterygoid muscle; *2.* swelling in the lateral pharynx; *3.* displacement of the tonsil and palate toward the median line of the mouth (see Fig. 1). With this displacement the tonsil may not be inflamed, thus differentiating a peritonsillar infection; *4.* swelling over the side of the neck often with swelling of the posterior border of the parotid gland.

Since the apex of the pharyngomaxillary space is at the level of the hyoid bone there is little or no swelling below the level of the angle of the jaw.

CONTENTS INVOLVED.

The internal jugular vein and the carotid vessels offer the greatest danger of complications.

One realizes the virulence, and extreme fetid odor, of suppurations near the air passages. The pus in the pharyngomaxillary space causes a rapid decomposition, and death of the walls of the carotid vessels before compensating hypertrophy can occur. This is constantly true of staphylococcus infections.[3]

The staphylococcus has a tendency to produce thrombosis of the blood vessels and lymphatics, and necrosis of the surrounding tissues, without lymphatic extensions as occurs with streptococcus.[3]

When the artery is attached there is apt to follow a profuse and fatal hemorrhage. When the jugular is attached there occurs much more frequently a thrombosis than a rupture and hemorrhage.

CASE REPORTS.

Case 1: Mrs. F. J., white, age 28 years, entered Christ Hospital on Jan. 7, 1935, with an acute follicular lateral pharyngitis. The tonsils had been cleanly removed three years before. Four days after entrance the right side of the neck showed diffuse external swelling, and at the same time the right lateral pharynx became swollen. Three days later redness of the right external ear occurred, and erysipelas spread rapidly over the right side of the neck and face. The temper-

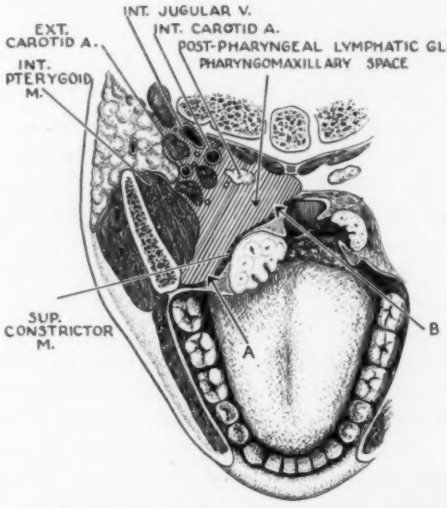

Fig. 1. Diagrammatic drawing of the right pharyngomaxillary space distended with pus. Note the distance of the major vessels from the sites of openings, A and B. The superior constrictor is a thin muscle.

ature ranged from 102° to 107° for one week with intermittent delirium. Antistreptococci serum was given repeatedly in large doses.

After one week the erysipelas showed signs of improvement, but the swelling of the neck was intense and the right lateral pharyngeal wall was pushed well toward the midline.

A diagnosis of a pharyngomaxillary infection with suppuration was made. In the presence of a cellulitis and tense external swelling of the neck, an incision was made fairly

high and just back of the right posterior pillar (see Fig. 1, B.). A curved tonsil hemostat was then inserted laterally and externally for a distance of two inches into a free cavity filled with pus. Drainage was maintained for one week and recovery progressed rapidly.

At this point I would like to mention that the husband, age 34 years, entered the hospital one week after the onset of his wife's illness. He, too, had a lateral pharyngitis, followed by erysipelas and a temperature of 107°. After a stormy course he recovered without complications.

Case 2: Male, age 28 years, who had had the right lower third molar extracted. The extraction was followed by trismus and a tense swelling under the angle of the jaw. The right tonsil, which was not undully swollen, together with the lateral pharynx, was displaced toward the midline.

A diagnosis of an acute suppuration in the right pharyngo-maxillary fossa was made, and incised as illustrated in Fig. 1, A. Following the incision a hemostat passed easily into the pharyngomaxillary space lateral to the tonsil and a large quantity of pus was obtained. Immediate improvement followed and the recovery was uneventful.

The purpose of this paper is not to minimize the importance of the external approach to the pharyngomaxillary fossa, but to emphasize the anatomical picture presented when this space is distended with pus (see Fig. 1), and the ease with which it can often be directly and simply approached through the mouth, at the maximum distance away from the great vessels.

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556 Doctors Building.

A FURTHER STUDY IN CLINICAL REPORT OF THE USE OF ACRI-VIOLET IN DISEASES OF THE UPPER RESPIRATORY TRACT AND EAR.*

DR. ARTHUR J. HERZIG, New York.

Since the publication of my article in THE LARYNGOSCOPE, June, 1926, "A Further Study and Clinical Report of the Use of Acri-Violet in Diseases of the Upper Respiratory Tract and Ear," occasion has been found to modify the type of cases the dye is used in, and same is presented to the medical profession.

Method of Preparing a Patient: The nose, postnasal space and pharynx, as well as larynx, are sprayed with a 2 per cent warmed bicarbonate solution. Then the same procedure is followed with a 2 per cent cocaine solution. The patient then rests for five minutes. The teeth, as well as the lips, are dried and rubbed with vaseline to protect them from being stained. Where the dye is applied to operative wounds the bicarbonate cleansing is omitted.

In applying acri-violet, or cocaine solution to the larynx and trachea, the tongue is held well forward and the patient instructed to inhale slowly. The spray is begun at the same time that the patient starts to inhale.

Acri-violet is only used in the strength of 1-200. It is compatible with iodoform and, therefore, the gauze may be moistened with the acri-violet solution, which is used for its astringent action. When the dye is used with plain gauze the dressings must be changed in 24 hours, but when used with iodoform gauze may be left as long as it is advisable to leave the iodoform gauze.

Acute Rhino-Pharyngo-Laryngo-Tracheitis (Grippe Colds): All membranes to be treated are sprayed with a 2 per cent cocaine solution. (Adrenalin is never used.) After taking effect, the postnasal space is cleansed with a mild alkaline solution. Mild suction is then applied to each nostril, thus

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producing a Bier's hyperemia and at the same time loosening any thick mucus that may be underneath the turbinate bodies and postnasal space. A warmed, mildly alkaline, postnasal irrigation is then given. The larynx is then sprayed with a similar solution, the patient holding his breath during this procedure. Acri-violet solution is then sprayed into the pharynx, larynx and trachea, the patient slowly inhaling, and should the patient fail to cough at the expiration of the spray, he is requested to do so as this coats the under surface of the vocal cords with the dye. The patient is given a prescription containing some of the cold tar products, as well as the salicylates, also a very small dose of extract of belladonna. Should the patient's cold continue more than 10 days, then the antra should be irrigated with a mild alkaline solution and the other sinuses inspected for infection. Codeine is prescribed should coughing become troublesome. The patient is instructed not to use his voice at all. Should temperature be present, the patient is advised to go to bed for 24 hours. This type of treatment is of the greatest importance in preventing sinusitis, or extension of the inflammation into the smaller bronchi.

Acute Follicular Tonsillitis: The pharynx is first cleansed with a mild alkaline spray, the cheesy masses in the crypts removed by means of a small curette and irrigated with a mild alkaline solution, (a special cannula being used for this).

Following this a drop of acri-violet is introduced into each and every crypt that is opened. Should any of the latter be closed, as is a common occurrence, a blunt-tipped probe is used to open same for similar treatment. Acri-violet is then sprayed into the pharynx. These patients receive a mild alkaline gargle for home use, as well as a solution of acri-violet. These home treatments are carried out every three hours until the patient's temperature drops to near normal. The patient is not confined to bed, but should remain in the house. Should there be any recurrence of cheesy material in the crypts the same aforementioned treatment is repeated. It is a common result to have a complete reduction of temperature within 24 hours, and only seldom is more than one treatment necessary. Should the patient request an icebag, allow them to use same. (Its only effect is psychological.)

Chronic Tonsillitis: Patients who are subject to the effects of chronically infected tonsils and are suffering from results

of the latter may be kept free of symptoms by periodic cleansing of the crypts, with a subsequent use of the acri-violet solution, after a suction cup is applied to each tonsil; this latter procedure is not employed in the acute forms. This type of treatment is only a temporary one, as nothing can replace a well performed radical tonsillectomy.

Vincent's Infection of the Throat: Whereas formerly the author depended upon acri-violet to kill the infection, he now uses it as a gargle, depending upon the intravenous use of neosalversan and local application of the latter to the infected areas. He has the blood examined for Vincent's, etc., before discharging any patient as cured.

Streptococcus Infection of the Throat, Larynx, Etc.: These cases are treated similarly to those of any other infection. The patient is confined to bed in all cases. Acri-violet is applied to all infiltrated tissues and sprayed into the larynx. A gargle of acri-violet is given to the patient to use as often as necessary. In severe cases with marked induration, the local treatment is augmented by injection of streptococcus vaccine.

Leukoplakia: It is interesting to note that when acri-violet is used as a gargle and mouth wash that the tongue appears clear and looks healthier after the dye is washed off. This does not seem to have any effect whatsoever on the course of the disease.

Laryngitis: After an alkaline cleansing, acri-violet is sprayed into the larynx (the patient inhaling at the same time). After the secretion has diminished, the spray should be changed to a milder astringent.

Tracheo-Bronchitis: The same treatment as enumerated above is followed.

Nasal Furunculosis: In these cases the use of acri-violet is almost specific in its action. During the 12 years that I have used the dye I have not had to incise a single case. The astringency, as well as the intense penetrating action of acri-violet, either aborts the infection — if the furuncle has not progressed too far — or facilitates a natural opening of the latter. After cleansing the skin gently with alcohol, and allowing same to dry, I apply a solution of acri-violet to the furuncle

and surrounding parts. I repeat the procedure a few times, always permitting the solution to dry before making another application. Gauze is then introduced into the nostril, having been saturated with the dye and the patient given a solution of acri-violet to apply with a dropper upon the gauze every three hours.

Furunculosis of the External Auditory Canal: In these cases the canal is painted with the acri-violet solution, after having been first thoroughly cleansed with alcohol. A slightly packed, plain gauze drain is saturated with the acri-violet and placed in the canal. The patient is instructed to moisten the gauze every three hours with the dye. A few of these cases have cleared without having had to resort to surgical intervention. Most of the furuncles, however, have had to be incised.

I have discontinued the use of acri-violet in antral infections, owing to its irritation.

Cervical Adenitis (Without Abscess Formation): The dye is applied to the pharynx and nasopharynx, and it is interesting to see how readily the adenitis responds to this treatment.

Operative Wounds: After all operations upon the nose and throat acri-violet is applied to the operated areas. All dressings, including the drain, or packing, in case of mastoiditis, as well as the semilunar gauze pad, which is placed directly upon the mastoid incision, are saturated with the acri-violet solution. The dressings must be changed daily. All radical sinus operations have the iodoform gauze saturated with the dye before filling the operated cavity with same. There seems to be less postoperative reaction than when the dye is omitted on the dressings. This is probably due to the marked astringent action. It is gratifying to know that there is a complete absence of the so-called typical mastoid odor in all cases where it is used. It also seems to stimulate healthy granulation tissue.

Acute Purulent Otitis Media: Acri-violet has been discontinued in these cases.

Chronic Purulent Otitis Media: The use of the dye has been discontinued in these cases.

SUMMARY.

Acri-violet has an intense bacteriostatic action on all types of infection, where the medication comes into actual contact with the diseased tissues. Where the infection is deeper, other methods must be employed.

CONCLUSIONS.

Acri-violet, employed in the dressings of operative wounds, exerts a stimulating action upon granulating tissue, and keeps the wounds clean.

667 Madison Avenue.

FOURTH INTERNATIONAL OTO-RHINO-LARYNGOLOGICAL CONGRESS.

The fourth International Congress of Oto-Rhino-Laryngology will be held in Amsterdam in 1940. The week from July 29 to Aug. 3 has provisionally been settled for the Congress.

A Committee of Honour has been appointed, composed of the professors of oto-laryngology in the Netherlands: C. E. Benjamins, Groningen; H. Burger, Amsterdam; P. H. G. van Gilse, Leiden; P. Th. L. Kan, Wassenaar; A. de Kleyn, Amsterdam; F. H. Quix, Utrecht; W. Schutter, Groningen; H. J. L. Struycken, Breda.

The Bureau of the Congress consists of the following members: Prof. H. Burger, Keizersgracht 317, Amsterdam C., President; Dr. A. A. J. H. Marres, Willemsparkweg 31, Amsterdam Z., Hon. Secretary; Dr. M. J. ten Cate, Obrechtstraat 55, Amsterdam Z., Hon. Treasurer.

A TRIO OF RARE BRONCHOSCOPIC CASES.*

DR. BERNARD WELT and DR. SAMUEL WEINSTEIN, Brooklyn.

The rarity of certain affections justifies the report of cases portraying those affections. These cases, in themselves, have a contributory value to the conglomerate experience of the entire medical profession. It is with this in mind that we have bethought ourselves of three unusual cases which we had the good fortune to see bronchoscopically within the past few years; namely, *1.* an endothelioma of the bronchus; *2.* a "benign inflammatory tumor" of the bronchus, and *3.* a web of the bronchus. We will report the cases in the order named above.

Case 1: No. 165,805, J. K., white, female, age 29 years, was admitted to the medical service of Dr. Joseph Rosenthal on Oct. 3, 1929.

Chief Complaint: Cough, 10 years' duration; epigastric oppression, three months' duration; hoarseness, three days; weakness and loss of weight.

Family History: Grandfather died of pulmonary tuberculosis. No contact.

Past History: Influenza in 1920; scarlet fever in 1923, ill for two months; no operations. Patient is unmarried.

Associated Tracts: Vomits once or twice before breakfast, and as a rule follows coughing spell; night sweats for 10 years; occasional hemoptysis during past 10 years.

Present Illness: Dates back to 1920, when she was confined to bed with influenza and left-sided pneumonia, also pleurisy. Fever and cough with pain over left chest. Advised after this illness to go to the mountains as a prophylactic for tuberculosis. The course has been one of remissions and exacerbations. Cough has been persistent, however, with production of thick, yellow sputum.

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*From the Otolaryngological Service of Dr. Philip Leibowitz, Jewish Hospital, Brooklyn.

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The past three months have shown an exacerbation of symptoms. Cough has become more violent, resulting in increase of vomiting. She feels feverish most of the day. Has lost 30 pounds in past three months. July 7, 1929, the patient went to Beth Moses Hospital, where thoracentesis was done and a diagnosis of interstitial fibrosis of the lung was made. Sept. 20, the patient went to Mt. Sinai Hospital, where diagnosis of benign tumor of left main bronchus was made, but no specimen was taken on account of hemorrhage. Hoarseness and some dysphagia has developed during the last three days.

Physical Examination: Adult female, appears chronically ill, although well nourished. Presents the following findings: Glands: Slight enlargement of anterior cervical, axillary and inguinal nodes. Trachea deviates and is fixed to left of midline. Left lung: Dullness anteroposteriorly; anteriorly to third intercostal space; posteriorly to sixth intercostal space. Absent breath sounds to fifth intercostal space on left posteriorly, but no rales. Heart displaced to left. Compensatory emphysema, right lung. Acute laryngitis.

Other Examinations: Temperature low grade, average 100.6°. Laboratory: Urine examination, negative; W. B. C., 9,800; polys., 63 per cent; monos., 35 per cent; Hb., 41 per cent; R. B. C., 3,700,000; basophils, 2 per cent. X-ray: G. I. series negative. Chest: Compensatory emphysema, right; obstructive atelectasis, left. Bronchoscopy (Oct. 12, 1929): Tracheal mucosa injected, thickened and covered with white purulent secretion. There is no distortion of tracheobronchial tree. At the level of the left upper lobe bronchial orifice a purple, partially lobulated tumor completely filling the lumen presented itself. The pedicle seemed to originate within the lumen of left upper lobe bronchus. By means of forceps the nodule was removed, accompanied by fairly profuse bleeding. The bronchial lumen distal to tumor appeared normal.

Pathological Report: Microscopic: Showed tissue to be the seat of a marked number of newly formed blood vessels, around which are found many uniform sized cells with deeply pink staining nuclei. There is no definite arrangement of these cells.

Diagnosis: Angioendothelioma. Nothing further was done for her, and against the advice of the hospital she was

discharged on Oct. 15. On readmission, on Feb. 6, 1930, it was seen that growth had taken place, and a radium capsule, 10 mg., was inserted, but with no subsequent effect. The X-ray examination showed nothing further than atelectasis of the left lung, but in view of the bronchoscopic findings it was felt that the entire picture was due to the obstructing tumor. She was discharged on Feb. 7.

In the interval she had submitted to a bronchoscopy in March, but no change had been noted except that the tumor nodule was slightly smaller. She was again bronchoscoped on this admission with findings similar to those of Oct. 12, 1929. No improvement was noted on readmission, June 6, 1930. The cough, with occasional hemoptysis, had persisted. Some dysphagia was complained of. There was no cachexia or loss of weight.

We lost sight of her until another admission on Jan. 4, 1933. No cachexia was noted. She appeared well nourished. Her complaint now was referred to the gastrointestinal tract, in addition to frontal headache. X-ray of chest revealed fluid occupying about 75 per cent of the thoracic cage. Examination of pleural fluid showed large, well formed endothelial cells, but no evidence of malignancy. The temperature ranged about 100°, occasionally going to 102°. She was discharged Feb. 5.

She was readmitted on Dec. 13 with symptoms of a severe bronchopneumonia. She died on Dec. 15. An autopsy was performed, of which the following is a resumé of the positive findings:

Autopsy 2,067: General: The body is that of a fairly well developed obese white female, age 32 years. Her hair is streaked with gray. She has a marked pogoniasis.

Cavities: Pleural: There are several broad, easily torn adhesions in the right pleural sac. No free fluid is present. The left visceral pleura is closely bound to the chest wall anteriorly and laterally. Medially, it is fastened to the pericardium by a thick mat of fibrous tissue. Posterolaterally, it forms the outer wall of a large cavity containing about 300 c.c. of creamy green pus. The inferior wall of this abscess is adherent to the left dome of the diaphragm. Pericardial cavity is obliterated by complete adhesion of the parietal and visceral layers of the pericardium. No pus or free fluid is found between the

two coats. The parietal pericardium is very closely adherent to the left mediastinal pleura.

Microscopic: Pericardium is thick, fibrous and edematous. Trachea is normal. Right bronchus is normal.

Lungs: The right lung is heavy, voluminous and covered by a dull fibrinous exudate. The right upper lobe and part of the right middle lobe are firm, not crepitant, and on section appear consolidated, gray-pink and mottled with dark gray, and slightly moist. The rest of the middle and entire right lobe are crepitant. Their cut surface is pink and gray, and the lung tissue is obviously air containing.

Pressure on the left lung *in situ* produces an abundant flow of creamy pus from the distal mouth of the severed trachea. The left lung presents a curious picture. It is almost entirely replaced by the large abscess mentioned previously. Only a narrow zone of lung tissue remains. This is in the hilar region and forms the superoanteromedial wall of the abscess cavity. It contains many small sacculations which communicate with the main cavity. The portion of lung lining the cavitation is very red and in places appears necrotic. From it dangle cords and shreds of dark red material. The remainder of the abscess wall consists of a thick coat of fibrous tissue that is firmly united to the pericardium anteromedially, the diaphragm below and the chest wall laterally.

In the left main bronchus is found a pink, soft, fragile ovoid mass, measuring 4x2½ cm. It completely obstructs the air passage. The tumor is attached to the bronchial wall, anterosuperiorly. On section it is fairly smooth, glistening and pale pink. Through its center a few white streaks radiate from the attached portion. In the posteromedial wall of the left main bronchus, just proximal to the tumor, is a tiny opening through which a probe may be passed into the adherent esophagus. Three anterior divisions of the left main bronchus are likewise occluded by pedunculated masses that seem to spring from the above described tumor. The posterior divisions of the bronchus are markedly congested. Every bronchus in the left lung is found to end in either the abscess cavity or the fibrous wall behind the pericardium. The former showed marked congestion and roughening of the mucosal surface.

Microscopic: The right lung shows extensive lobar pneumonia in its upper and middle lobes in the stage of gray hepatization. The rest of the lung shows areas of congestion, edema and emphysema. The pleural covering is thick, fibrous and bears a superficial fibrinous exudate. Some areas of the right lower lobe are densely packed with polys. Other areas contain distended alveoli, in which there are a few desquamated epithelial cells and a small number of polys.

The left lung is virtually a mass of fibrous tissue, containing very thick blood vessels, and large collections of round and plasma cells. Many tubular structures lined by cuboidal cells

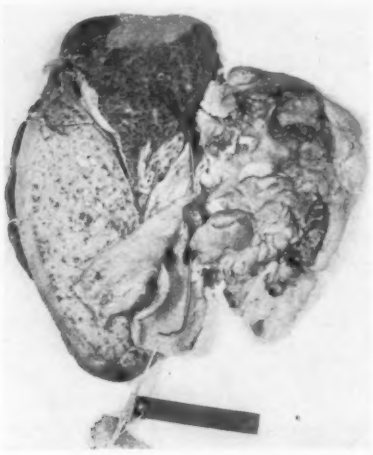

Fig. 1. Case 1. Gross specimen. Probe in tracheoesophageal fistula.

are seen throughout the section. A number of these are obviously bronchial glands. There is a peribronchial round cell infiltration and considerable anthracosis. One portion displays a granulomatous formation having a multitude of plasma cells enmeshed in strands of fibrous tissue, which also contain numerous fibroblasts, many newly formed blood capillaries and several foreign body giant cells. An adjacent area shows a large number of polys.

The mass is made up of fibrous tissue trabeculae and tumor cells. The ramifications of the traebeculae do not break up into fine fibrils, but remain relatively thick, so that there is

no close intermingling of individual tumor cells and fibrous framework. Instead there are definite and distinct bands of fibrous tissue and equally definite and distinct islands and cords of tumor cells. The former are composed of numerous mature fibrocytes and connective tissue fibres, which stain a brilliant red by the Van Gieson method. Fine blood spaces are seen in practically all the septa. In fact, a great many of the septa appear to be nothing more than small and medium sized blood vessels that run between agglomerations of tumor cells. This formation is in some places so striking that the tumor gives the appearance of an arborization of blood vessels with the spaces between them occupied by neoplastic cells. These spaces vary in size and are mostly irregular, but they are accurately filled by tumor cells. Thus, where the vessels are widely separated, the interspaces contain sheets of tumor tissue whose outlines conform to those of the vessel walls. Where the vessels are closer together the cells lie not in sheets, but in cords one or more layers deep.

Many parts show a honeycomb structure, with each septum or blood vessel separated from its nearest neighbor by a mantle or partition of tumor cells which is at least one layer thick. In places the tumor pattern bears a resemblance to lung parenchyma. The artificial alveolar walls are made up of double rows of tumor cells two layers thick, and the artificial alveolar spaces are occupied by loose fibrous tissue and a few blood capillaries. In some places there is a diffuse distribution of tumor cells, rather than a formation of cords or sheets of cells. The tumor cells extend up at one point to the very surface of the neoplastic mass and here are confined by only a thin lining of flattened bronchial epithelium.

The tumor cells are small, round, cuboidal and short columnar with indistinct cell membrane, and fairly abundant, granular well stained cytoplasm. Their nuclei are moderate in size and centrally placed. They have a distinct nuclear membrane and coarse, darkly stained chromatin particles. Many nuclei are pycnotic. No mitotic figures are seen. There is no evidence of invasions of the many blood vessels by their surrounding neoplastic cells. However, at the periphery of the section, these cells show a tendency to unrestrained growth into the subepithelial tissue. A lymphatic space is partly filled with tumor cells. A small bronchus shows its

mucosa replaced by fibrous tissue with round and plasma cell infiltration.

Tracheobronchial glands show replacement of the lymph sinuses by tumor tissue, and in the region of left upper lobe bronchus there are two nodes of tumor tissue continuous with the growth inside its lumen.

Esophagus is normal, except for a small portion at the level of the main left bronchus. At this point there is a fistulous

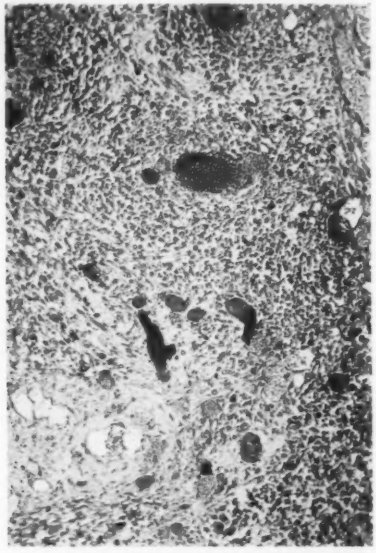

Fig. 1. Case 2. Photomicrograph of biopsy specimen from uterus. Myogenic sarcoma.

communication with the latter structure. Following is summary of autopsy findings:

Anatomic Diagnosis: *1.* Perithelioma of left bronchus; *2.* pulmonary abscess, left; *3.* lobar pneumonia, right; *4.* fibrous pericarditis; *5.* fibrous pleuritis, left and right; *6.* infectious splenomegaly; *7.* focal nephrosclerosis; *8.* bronchoesophageal fistula; *9.* fibromyoma uteri; *10.* obesity; *11.* hirsutism; *12.* bacteriology: Nonhemolytic streptococci

and B. coli from pericardium and lung pus. (Autopsy No. 2,067; Hospital No. 123,558.)

Primary endothelioma of the bronchus is a rare affection. According to Banyai and Grill,[1] most authors believe that it originates in the pleura and secondarily infiltrates the lung diffusely. With regard to the case which we are reporting we are unable to say whether the tumor of the bronchus originated either in the pleura or the lung, but other sources were excluded by a complete autopsy.

Ewing[2] says the term "endotheliomata" is applied to an extensive group of tumors which are believed to be derived from the lining cells of blood vessels, lymph vessels and spaces, subdural spaces and serous cavities.

Kaufmann[3] in discussing tumors of the pleura says: "Endotheliomas are rather rare. They are derived *a.* from the endothelium of lymphatics and clefts (lymphangioendotheliomas), and *b.* from the lining cells. The author believes in both. According to the author's experience, it is easier to conceive of the latter than the former."

Broden,[4] reporting a case of endothelioma of the lung, says an endothelioma is a tumor derived from endothelial cells. Hence it may arise from the lining cells of blood vessels, lymph vessels and spaces and serous cavities. While the definition is simple there are the very widest of diverse opinions as to just what tumors should be classified under the term "endothelioma."

In this respect Delafield and Prudden[5] say that no class of new growths has given rise to so much controversy and remained so indefinite as this one. There is, in fact, hardly an obscure tumor that has not at one time or another since the appearance of Volkman's monograph been called an endothelioma.

In respect to the identification of endothelioma we want to quote Prof. D. A. Welsh[6] rather fully:

"New growths of endothelium are often hard to classify. The best way is to regard endothelium as having a close structural analogy with epithelium, and to recognize that there are *1.* surface forms of endothelium (analogous to surface epithelium) lining the larger serous cavities, like the pleura, the

membranes of the brain, the lining of the joints, etc., and *2.* tubular forms of endothelium (analogous to glandular epithelium) lining the blood and lymph vessels and sinuses. From each of these forms of endothelium there take origin simple and malignant new growths analogous to the corresponding epithelial new growths, so that there is also a close biological analogy between these two types of new growth.

"But a difficulty arises from the fact that endothelium is less stable and less constant tissue than epithelium so that its new growths are not always true to type. Thus some endotheliomata which take origin from blood or lymph vessels may revert to a connective tissue type of structure, that is, some

Fig. 2. Case 1. Photomicrograph of biopsy specimen from bronchus. Endothelioma.

new growths of endothelial cells develop a fibromatous or sarcomatous structure. Thus arise the fibroendothelioma, the angiosarcoma, etc., which rarely have any epithelial analogue.

"Hence two facts are essential to a proper understanding of the endothelial new growths: *1.* The endothelial cell is the dominant and provocative element in a new growth, whether simple or malignant, just as certainly as the epithelial cell is the dominant and provocative element in an epithelial new growth. The fact is recognized in the more malignant types (endothelioma), but it is ignored in the less malignant types (angioma). The true analogy between angioma and adenoma

has been overlooked. *2.* The endothelial cell is morphologically less stable and less constant to type than the epithelial cell, so that while a fibroblastic epithelial reaction is rare, a fibroblastic endothelial reaction is common. In many forms of endothelioma it is the rule, and not the exception, for certain endothelial cells to become fibroblastic and to take part in the formation of the stroma of the endothelial new growth. That happens regularly in the endothelioma of the pleura, for example, and in many other forms."

Now there are several comments which we wish to make in reference to this case. It will be noticed that the patient dated her illness back to an attack of influenza in 1920. Banyai and Grill, in reporting their case, also noticed that many instances of pulmonary malignant tumor are described with an acute onset and are accompanied by pain in the chest or induced by a febrile attack. Patients with early symptomless lung tumors may become conscious of their pulmonary disease following influenza or other severe disease of the lungs because such a disease may increase the bronchial stenosis already present to a symptom producing degree.

In the second place, this case was observed over a number of years and so this is evidently a slowly developing process. The thought, therefore, occurs to us that when one has the opportunity to make the diagnosis of endothelioma of the lung in the early stages of a case, whether the surgeon should not be called into consultation for the purpose of performing a pneumonectomy. Perhaps this patient might be alive today had that procedure been followed.

Case 2: No. 192,352, Mrs. S. W., age 58 years, Hebrew, widow, was admitted to the gynecological service of Dr. Schwartz and Dr. Tran on Jan. 11, 1936. She came into the Jewish Hospital primarily as a gynecological case, and we will therefore make the resumé of this case as brief as possible, trying to state only that which is pertinent to our interests.

She came into the hospital complaining of vaginal bleeding of one week's duration. In the past history we find that she had "bronchitis" for the past six or seven winters, that is, for the past six or seven years she has had pains in the chest with cough every winter. Although she didn't know her exact

weight, she felt that she had been losing weight for several months as her clothes had become loose.

She had been in apparent good health until two or three months before admission, when she developed frequency of urination and nocturia, and burning on urination. For two months prior to admission she noticed that she became easily fatigued and that she had increasing weakness.

During the entire winter she had a cough, which developed into a cold with fever lasting five days, and she became free of the fever two days prior to admission.

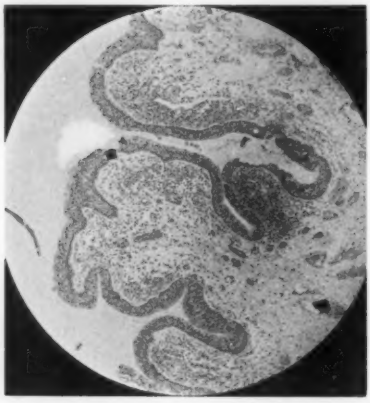

Fig. 2. Case 2. Photomicrograph of biopsy specimen bronchus. "Benign inflammatory tumor."

One week prior to admission she developed vaginal staining which became quite profuse, and was accompanied by lower abdominal cramps on the day prior to admission.

The pertinent facts in the physical examination on admission are as follows: Lungs: Decreased breath sounds and occasional moist rales at left base posteriorly. Increased dullness over same area. Abdomen: Relaxed, firm mass reaching centrally to the umbilicus. Vaginal: Parous introitus, elongated, bluish soft mass occupies the vagina; when traced is seen to be coming through markedly dilated cervical canal. Mass bleeds readily; foul odor. Uterus enlarged to umbilicus;

hard and smooth. The impression was fibromyoma of the uterus with submucous degenerated fibroid.

The patient on Jan. 13 "delivered" a "fibroid" from the vagina. She continued to cough, having a low-grade temperature, ranging between 99° F. and 100° F. She brought up thick, tenacious phlegm.

The pathological report on the spontaneously delivered "submucous fibroid" was: Sarcoma, myogenic of uterus (Dr. Bela Halpert). X-rays of the chest on Jan. 16, did not reveal any characteristic pathology.

On Jan. 21, she was seen in medical consultation by Dr. Eis, who reported: "Marked diminution of breath sounds at the left base posteriorly and a corresponding decrease in tactile fremitus. Many varied rales are obtained at both bases and throughout left lung. At right base the breath sounds are even exaggerative. Advise restudy of chest by X-ray; sputum examination for Koch and malignant cells; passive bronchography and finally bronchoscopy, if necessary. One is not justified in excluding malignant metastases, even in spite of former negative X-ray. I would also advise X-ray examination of necks of femora."

Restudy of the chest by X-rays and of the femora and pelvic bones did not reveal any localized pathology which could be interpreted as metastases.

The cough and hemorrhagic vaginal discharge continued. Sputum examination on Jan. 21, 23 and 25 did not reveal any tubercle bacilli, nor cells indicating malignancy. Blood, Kline test, negative.

We saw the case in consultation on Jan. 21 and performed a bronchoscopy on Feb. 4. Bronchoscopic report: Local anesthesia, cocaine 10 per cent. No. 7 bronchoscope passed. Right bronchi congested. A pale pink neoplasm is noted in the lumen of the left lower lobe bronchus. Bleeds easily to the touch and has a benign appearance. Bronchi congested and landmarks obliterated. Advise passive bronchography.

Diagnosis: Bronchial neoplasm of left lower lobe bronchus. Biopsy taken.

On Feb. 11 a diagnostic D & C had been done. We might mention here that the pathology laboratory reported the curet-

tings as sarcoma, myogenic of the uterus (Feb. 19). The patient continued to cough considerably and the chest signs remained unchanged.

We rebronchoscoped her on Feb. 13: No. 9 bronchoscope used. Marked purulent discharge from the left main bronchus. Neoplasm noted in the lower part of the left bronchus at about the bifurcation into the upper and lower lobe. Pedicle difficulty made out; four pieces taken for biopsy. Suggest bronchoscopy in one week.

We will here give the pathologic reports in detail on the specimens taken in this and the previous bronchoscopy.

Specimen taken Feb. 4: *Description:* Gross: The specimen consists of three fragments of firm tissue up to 0.6x0.4 cm. All of them are taken for microscopic study. Microscopic: One surface of this fragment is covered by pseudostratified cylindrical epithelium which rests on a basement membrane. The tunica propria is loose and infiltrated with small round cells and large mononuclear cells. This infiltration continues into the intermuscular tissue. Beneath this there is a fragment of cartilage with many blue granules in the ground substance and calcification of some of the cells. In another fragment groups of acini of mucous and serous glands and their ducts are seen. No structure resembling neoplasm is seen.

Diagnosis: Bronchial wall, fragments of, with chronic inflammatory reaction.

Specimen taken on Feb. 13: *Description:* Gross: The specimen consists of five fragments of soft white tissue measuring up to 0.5x0.3x0.2 cm. All of these are taken for microscopic study. Microscopic: Serial sections from the fragments show one surface covered by pseudostratified cylindrical epithelium. This rests on a basement membrane, beneath which there is a loose vascular fibrous connective tissue densely infiltrated with small round cells and large mononuclear cells. Many of the latter are eosinophiles. The surface of another fragment is thrown into delicate folds. The subepithelial layer shows similar structures as those described. In a third fragment the mucosa and muscularis mucosae are noted, beneath which there are groups of acini of mucous and serous glands. The lumina of some of these are full of secretion. The connec-

tive tissue is infiltrated with small round cells and large mononuclear cells.

Diagnosis: Bronchial mucosa, fragments of, with chronic inflammatory reaction.

She was bronchoscoped again by us on Feb. 20. Report: No. 9 bronchoscopic tube used. Left main bronchus lumen occluded by two polypoid masses associated with a moderate amount of purulent discharge. These masses were removed with a snare, and lumen was restored. Biopsy sent to laboratory. On the following day because of the presence of inguinal glands, the gynecological service deemed it preferable to treat this patient with deep X-ray therapy and radium instead of by surgical intervention (*i. e.*, for uterine condition). She was accepted for X-ray therapy by Dr. Wasch, the radiologist of the hospital. She was discharged from the hospital on Feb. 24 and she came to the X-ray department for three months for therapy.

The following is the pathological report on the bronchoscopic specimen removed Feb. 20: *Description:* Gross: The specimen consists of two portions of firm tissue 1.5x0.5x0.3 and 0.7x0.5x0.4 cm., respectively. Their external surfaces are red-brown and slightly nodular. The cut surfaces are made up of red-brown tissue in which circumscribed yellow areas are present. All of the smaller nodule and a longitudinal section through the larger are taken for microscopic study. Microscopic: A part of one surface is partly covered by stratified squamous epithelium, partly denuded, and partly covered by pseudostratified cylindrical epithelium, and is thrown into coarse, low folds. The capillaries in the tunica propria are distended with blood. In the loose supporting connective tissue there is a dense infiltration with small round cells, large mononuclear cells and polymorphonuclear leukocytes. The denuded surface is covered by fibrin and extravasated blood, which in the deeper layers shows a number of fibroblasts. Another fragment is composed almost entirely of vascular granulation tissue densely infiltrated with small round cells, large mononuclear cells, and polymorphonuclear leukocytes. In the submucosa there are groups of acini of serous and mucous glands and their ducts.

Diagnosis: Bronchial mucosa, portion, with chronic and acute inflammatory reaction.

The patient was treated in the X-ray department three times a week for about three months, when she again had a recurrence of vaginal bleeding and abdominal mass. She was readmitted to the gynecological service on May 24. At this time, as she was in an inoperable condition, and as she did not respond to X-ray therapy, she was transferred to the Kings County Hospital.

On her second admission the patient had no complaints in reference to the chest. On physical examination the lungs were resonant to percussion, and the breath sounds showed a rather prolonged and harsh expiration with a few squeaks.

We communicated with the Kings County Hospital and were informed that the patient demanded her release and was discharged against advice the day following her admission. We do not know of the further progress or outcome.

This case is especially interesting because of the two conditions present in the one patient, namely, a myogenic sarcoma of the uterus, and a "benign inflammatory tumor" of the bronchus.

The gynecologists and the internists naturally were first inclined to consider the pulmonary complaints as due to a metastatic lesion from the uterus. This we were able to rule out by repeated bronchoscopic biopsy.

We are not concerned here with the sarcoma of the uterus; for us it is purely incidental, though interesting to be sure.

We shall now make several comments in reference to "benign inflammatory tumor," a designation bestowed upon these lesions by the Jacksons. As the Jacksons[7] have pointed out, the fact that these tumor-like inflammatory hyperplasias are histologically not true neoplasms does not detract from their great clinical importance, for when a bronchus is obstructed by a tumor, no matter what its histological nomenclature, the defensive power of the tributary portion of lung is lowered, secondary pathologic changes (atelectasis, suppuration, pulmonary abscess and bronchiectasis) are almost certain to follow, and the seriousness of these consequences may be out of all proportion to the innocence *per se* of the primary lesion, if it had not been obstructive.

Jackson thinks that stagnation of secretions and infection may be etiologic factors, but this is not definite.

Two symptoms which are claimed to be almost always present, namely, *1.* wheezing heard at the open mouth, and *2.* hemoptysis, were not present in our patient.

Diagnosis is, of course, as in this case, by bronchoscopic biopsy, and treatment is the removal of the entire growth bronchoscopically, so as to restore perfect ventilation and prevent suppuration in the tributary portion of the lung. If they are not discovered and removed they produce pulmonary complications which result in the death of the patient.

It is furthermore very essential to remove these tumors, because Wessler and Rabin[8] claim that they have definite evidence that a benign bronchial tumor may years later undergo malignant degeneration.

Peroni,[9] in speaking of these "inflammatory tumors," says that these lesions are tumors in the physical sense of tumefactions, not in the sense of blastomas, and are inflammatory because inflammation is their most probable origin, or at least best explains their histologic and clinical character.

He says, further, they are undoubtedly tumors from the clinical point of view, because they tumesce into the bronchial lumen, but he does not mean by the tumors that they are blastomas, because they have no sign of atypical proliferation.

Because of their gross and morphologic characteristics, and until pathologists have reached an accord on the denomination of these tumor-like inflammatory lesions of the respiratory tract, he believes that for the present the only rational denomination with which to qualify the gross appearance of the growths and their probable origin is inflammatory polyps of the bronchi.

Case 3: No. 187,190, M. R., age 32 years, white, American-Hebrew, dentist, was admitted to the medical service of Dr. M. Rabinowitz on Jan. 10, 1936, because of paroxysms of coughing present for the past six years.

Family History: Negative. *Past History:* Negative for diphtheria as a child; frequent sore throats and tonsillitis. Present illness began six years ago during the latter part of the summer, when he developed what was called whooping cough. At this time he had paroxysms of coughing, with

inspiratory gasps, which terminated with a vomiting spell. The cough was worse in the morning on arising from bed, and on lying down at night. X-rays of the chest were negative.

The cough disappeared after four months, only to return eight months later with similar symptoms. X-rays and sputum examinations again were negative. He was told his symptoms were due to bronchitis. This episode lasted three months, creosote and codein being ineffectual in producing relief. He had a third attack again in September of the next summer, and a fourth attack with the appearance of winter and the first snowfall. At this time he had a cold with moderate temperature. In the past six years he had 10 attacks.

The attack at the time of admission began on Dec. 20, 1935, following a sore throat and grippe, with fever of 103-104° F. He rarely expectorates, never has hemoptysis, but he has had night sweats. He had himself cupped by a barber several weeks before because of continued pain in upper chest on coughing. He was referred to the hospital for bronchoscopic examination to rule out the presence of bronchial polyp or foreign body aspirated at some unknown time.

Examination: Temperature 99° F. Not acutely ill. Well developed and nourished. Swarthy complexioned with slight suggestion of cyanosis of fingers. Tonsils are buried, cryptic and injected. Lungs: Tactile fremitus slightly increased at the right base where slight amphoric breathing sounds and occasional coarse rales and rhonchi are heard. No friction rub heard. Lipoma present on right thigh. Several small hemorrhoids present.

Impression (of medical service): Chronic bronchitis with possible bronchiectasis at right base. Chronically infected tonsils. Urine normal. White blood cells, 8,950; polys., 52 per cent; lymphs., 48 per cent. Red blood cells, 4,530,000; hemoglobin, 90 per cent. Platelets, 290,000. Sedimentation rate, 31 mm. (clear serum). Blood, Kline test, negative.

Sputum examination (Jan. 16, 1936): No Charcot-Leyden crystals; no Curschman spirals; no blood; no elastic fibres; smear and antiformin, no tubercle bacilli.

Throat culture (Jan. 19): Staphylococcus aureus, streptococcus nonhemolyticus, streptococcus viridans.

Sputum culture (Jan. 15): Micrococcus catarrhalis; staph. albus; strept. hemolyticus; strept. viridans; strept. nonhemolyticus.

On Jan. 14 Dr. S. J. Cohen made the following note: A six-year history of paroxysmal coughs. The physical examination is negative except for a few coarse rales on the right side. The history also brought out the fact that the cough is often produced by the recumbent posture. The possibility of a foreign body or bronchial polyp must be considered. The nature of the sputum (mucopurulent) and the chest signs speak for bronchiectasis. The patient was seen in consultation by us on Jan. 15 and bronchoscopy was advised.

X-rays of chest (Jan. 16): The aortic arch is somewhat dilated. The cardiac shadow is apparently within normal limits of size, shape and position. There is a dense bilateral hilum infiltration with exaggeration of the pulmonary markings at the bases. A lipiodol study would be advisable to determine the nature of the terminal bronchioles.

Bronchoscopy (Jan. 16): Findings and procedure: Larynx shows interarytenoid thickening which is inflammatory in character. The trachea and bronchi show a marked hyperemia. The right bronchus presents a gray, glistening membrane attached to the bronchial wall in part. A piece was taken for biopsy.

Diagnosis: Bronchial web. Suggest bronchography and subsequent bronchoscopy.

A passive bronchography was performed by the X-ray department on Jan. 17: Report: A lipiodol study reveals a slight degree of dilatation of the terminal bronchioles on the left as compared to the right.

Pathological report on bronchoscopic biopsy: *Description:* Gross: The specimen consists of a fragment of gray-white tissue 0.7x0.2 cm. All of it is taken for microscopic study. Microscopic: Part of one surface is covered by pseudostratified cylindrical epithelium. In the tunica propria there is an infiltration with small round cells, large mononuclear cells, and some polymorphonuclear leukocytes. A few strands of smooth muscle bundles are seen separated by vascular fibrous connective tissue. No atypical proliferation of the epithelium is seen.

Diagnosis: Bronchial mucosa, portion of, with chronic inflammatory reaction.

The patient was discharged on Jan. 20 in the care of Dr. S. J. Cohen, who was his family physician. Upon recent communication with Dr. Cohen we learned that the patient is doing well and that he has no complaints. This condition may be more common than a paucity of the literature may indicate, but it certainly is not very frequent.

The Jacksons[10] speak of congenital web of the bronchus and say that in the three cases which they saw at the Bronchoscopic Clinic the patients came because of suspicion of foreign body. It will be remembered that foreign body was suspected in our case. They[11] say that the bronchoscope affords the only certain means of diagnosis and that bronchoscopic dilatation will cure most cases if used in infancy and early childhood.

We do not believe that our case is one of congenital web of the bronchus. It, no doubt, developed in later life as the result of some inflammatory process. In this respect, the Jacksons[12] tell us that, "We have observed quite a number of cases of congenital web of the bronchus. Additionally, we have seen many other cases where the web seemed to have developed as the result of pathology in later life."

We punched out the web in our case, and the patient has apparently been cured.

CONCLUSIONS.

1. We have presented three interesting and unusual types of bronchoscopic cases, namely, *1.* endothelioma of the bronchus; *2.* benign inflammatory tumor of the bronchus, and *3.* bronchial web.

2. The value of bronchoscopy in obscure chest conditions is again demonstrated.

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THE EARLY STORY OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY BY ONE OF ITS FOUNDERS.

DR. DWIGHT L. HUBBARD, Denver.

To be lifted bodily and suddenly carried 2,000 miles and put down in surroundings of nearly 50 years ago would be no stranger than the events of the past few days during the Forty-second Annual Meeting of the American Laryngological, Rhinological and Otological Society, which gathered in Denver on May 18-20, 1936. At this meeting I rejoiced to see my old friend and colleague, Dr. Thomas J. Harris, who was present as the guest of honor. At the first day's meeting Dr. Harris presented a scholarly resume of the history of laryngology with special reference to the history of this Society from its inception to the present time. During the reading of his paper Dr. Harris very graciously introduced the writer of this article to the Society as one of its three living founders. Does anyone wonder that I should feel so vividly the lift of the magic carpet as I was transported back to the scenes of those days of 1895 in faraway New York, then and for 40 years thereafter, my home and workshop? Parenthetically, I am happy to say that after these years of strenuous work in that difficult field, I found a lovely haven for resting in beautiful Denver. All ye weary ones, tired of the constant urge for more and better things, come out to Colorado where you will be surprised to find the more and better things are here ahead of you.

Apropos to the visit enjoyed with Dr. Harris, it is needless to say that conversation was directed into channels leading to the beginnings of this Society. Reminiscently, we were led to talk of those who have gone before and of the work they did, not only privately but of their earnest endeavors in their special fields. It occurred to us that I am the only living member of the group of founders who has an intimate knowledge of the events leading to this organization. It does not seem best to ask either of the other two remaining mem-

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bers who took part in the formation of the Society to attempt the recital of these events, one on account of advanced age and the other because of lack of knowledge of the necessary sequences, and not because of any lack of interest in the matter. So I undertake the task with a proper appreciation of its responsibilities as to accuracy and interest.

It is a difficult task to put down plain, dry facts and make the matter readable; nor would it be good history if along with these facts a recital of some collateral points of interest should not be given. I shall, therefore, take the liberty of going somewhat aside from the path of plain incident and try to make the reading both interesting and useful as a matter of recorded history. The weight of an obligation is upon me, because I believe it is important that the true status of our Society should be claimed and made known abroad, for I feel keenly that the advancement that has been made in laryngology, rhinology and otology would not have been accomplished without the foundation that was laid in these early days of which I am writing. Important happenings are so soon forgotten. I am reminded of a remark made by Dr. Harris during our conversation when I told him I had some hesitation in the writing of these archives because of the necessity of the use of the personal pronoun, to which he replied, "It will not be long before both you and I will be forgotten, but the record will stand." So I will boldly perform my duty with, I hope, becoming modesty, stating much of what I can remember that is not now a part of recorded history. I cannot ask for corroboration, for there is no source of which I have knowledge.

It will be necessary to go back into the dim past for a fragment of information gained from others to establish connecting links in the formation of the chain which led to the establishment of the three-in-one idea. I gained this information personally from our old mentor and consultant, Dr. O. B. Douglas. It was somewhere about the year 1880 that a small clinic was established in East 34th street in a modest English basement house for the purpose of giving aid to the poor of the east side in diseases of the eye, ear and throat. As far as can be learned, the names of the men who established this clinic are as follows: David Webster, who became a recognized authority on both eye and ear; D. B. St. John Roosa, an international authority on the eye and a widely

read author on the subject; and O. B. Douglas, a Christian gentleman and most conscientious laryngologist, who became one of the most important influences toward the coming together of the men of the ear and the throat. From this beginning developed the Manhattan Eye and Ear Hospital, with, for some unknown reason, emphasis placed upon the eye and ear. I do not know the date of its removal to the site at 41st street and Park avenue. I am certain, however, that it was in this building that the foundations of our Society were laid. It was to be quite a good many years before a close fraternal relationship was to be established between ear men and throat and nose men. My own intimate connection with the clinical work in the throat department began about this time, so from now on this account will be a matter of personal observation and experience.

It was not destined to be all easy sailing, for, like all new developments, there was a variety of opinions as to policy and conduct. The eye department was placed in one end of the building on an upper floor, the ear department in the other end of the building, and the throat department on the ground floor back of the executive office. This was fortunate, for we had the strategic position, easily accessible and comfortable. A common operating room was on an upper floor. A few years afterward we turned out the *nerve department* and took over that room for an operating room for our throat cases. And what was a nerve department doing in an eye, ear and throat hospital? Politics, of course.

It may be surmised from what has been already said that all was not as harmonious as should have been the case; then came some changes for the better. It was largely due to the kindly influence and diplomacy of Dr. Douglas that the atmosphere began to clear, and the hospital became the Manhattan Eye, Ear and Throat Hospital. From now on I will more particularly dwell on the throat and ear aspect of the hospital, as it is that department which most deeply concerns us.

Now began to appear that hydraheaded monster of all clinical hospitals, the abuse of clinical material. Established primarily for treatment of the poor, it began to be evident that patients were coming who were abundantly able to pay a private physician some kind of a fee. My intimate associa-

tion with Dr. T. Passmore Berens of our staff (one of the founders of our Society) led to the establishment of the first Night Throat Clinic in the city. We had abundant success from its beginning. Our idea was that more worthy clinical material would take advantage of a night clinic than of the day sessions, which soon proved to be true. An Ear Clinic followed, and then came the uniting flux. This is the true story and I will not make it any better than it deserves: It so happened that the Grand Union Hotel was on the next corner from the hospital, and Mr. Simeon Ford, among many other attractive features of a good hostelry, maintained a very comfortable grill room in which were served at convenient hours for tired clinical assistants and assistant surgeons, pilsener and pretzels, or whatever else the tired ones desired.

To this haven we would frequently retire and while partaking of ice cream, soda or lemonade, we naturally drifted into such companionable conversation as we found most profitable. And thus the work at the hospital came into consideration. These small conventions were generally held in the evening after the clinic, when we could gather the men from the ear clinic with our own throat men, making quite a respectable representation of the hospital. I am quite sure that the interdependence between ear and throat had received quite a boost at our hands during these conferences. I am not claiming originality for the idea, for it had been growing elsewhere, but the growing importance of the Manhattan Eye, Ear and Throat Hospital gave added importance to our influence. An occasional visitor from other clinics would join us at our evening clinic, sometimes out of curiosity to see how the evening idea was thriving, but later with interest in our discussions at the rendezvous. The community feeling was growing.

Now comes another factor: The then very young Post-Graduate Hospital was struggling in its birth pangs in East 20th street in a two-story brick building and welcomed any assistance it could obtain. My associate, Dr. Berens, and I had no difficulty in being appointed instructors there in the afternoon clinic. This brought us in contact with men from out of town who came to learn the rudiments. We gave them a cordial invitation to visit our evening clinic, and the opportunity was avidly accepted. These men were made welcom- also in the Ear Clinic. As our evenings grew apace, so our

instruction became popular. The combination was working well.

I used the expression above "to learn the rudiments." Indeed, it was just the rudiments that appeared to be necessary. It would be supposed that a practicing physician who came to the city to study a specialty would have a smattering idea of the work. It was rare that we found any who knew how to adjust and manipulate a head mirror. At the Post-Graduate Hospital in those early days the instruction received was largely visual over the shoulder of the instructor, which was little enough. Six weeks of this instruction made a "course." Little satisfactory work could be done in this manner, and this was our chief reason for inviting a select few to come to our evening clinics for more personal and satisfactory work. Those accepting our invitation made rapid progress.

I must give one interesting feature of our teaching, which will be illustrative of the whole, although in general the cases did not have such a dramatic aspect. This patient was a short, widely-built, stocky and muscular Italian; very good-natured and willing to do what was requested of him. We called him Svengali. He spoke very little English, but understood very well, and being very intelligent and accommodating, made an ideal patient. He had the widest throat and the largest larynx I ever saw. Coming to the clinic for a hoarseness due to his occupation as a vendor on the street, we soon discovered that he had a most unusually tolerant larynx. A very slight local anesthesia sufficed to permit almost any kind of manipulation of the larynx, allowing us to demonstrate the technique of making applications, making a study of the different movements of the vocal bands over which he had great control and giving a splendid opportunity for study of the entire trachea. The individual cartilages could be easily seen and counted down to the bifurcation. He would tolerate a piece of cork the size of a small pea, attached to a silk thread, dropped into the trachea, when the forceps would be introduced and the cork extracted. Svengali not only enjoyed his popularity, but was always grateful for the silver offering he would generally receive. I speak of this remarkable case to illustrate our method of gaining a little scientific recreation and much useful information from the material at hand.

I think our clinic was one of the first, if not the first, to make judicious use of the galvano-cautery. We had no electricity in the hospital, making the use of storage batteries necessary. I remember well the first introduction of the galvano-cautery apparatus. Now we would have means to stop the unscientific treatment of hyperplastic and hypertrophied turbinates by means of either surgical removal or by application of chromic, or mono- or tri-chloracetic acid. It is my belief that we saved many of these cases from a future dry condition which would have been almost inevitable. Never the flat blade; always the fine cutting edge of the platinum knife! We used platinum in those days for it did not cost a king's ransom. I speak of this to show how we had to make the best of the material at our command. It was quite a contrast with the modernly equipped hospital or office. In later years I was to become well acquainted with modern equipment. We were to see many new things in those days that are old now, but we certainly had opportunity for study that was rare, although much of it in the pioneering stage. The important question is: Did we blaze a good trail that others could follow? It would seem to me that the present status of this Society would warrant the assumption.

It would be unjust and would seem ungrateful if acknowledgment of the valuable services of our collaborators were not made by mentioning the names of those who helped in the lead of the movement. It will be impossible to mention more than a few, for the list grew and grew until the few of the beginning became the cohorts composing the membership of the American Laryngological, Rhinological and Otological Society. The order is given at random as the names are suggested. These men represent several different hospitals, clinics and civic organizations, both public and private.

Dr. David Webster, Dr. Roosa, Dr. Emerson, Dr. Neil J. Hepburn, Dr. Mathias L. Foster, Dr. O. B. Douglas, Dr. Berens, Dr. Haskins, Dr. Coffin, Dr. Myles, Dr. Nichols, Dr. Harris, Dr. Phillips, Dr. O'Dwyer and several others just as worthy of mention. I think only about four of these are still living, but they have left behind them a lasting influence for good and for progress.

It should be a matter of interest and personal pride for us to consider a few of the outstanding achievements of our brethren, some of which were revolutionary, leaving a stamp

of permanency on the history of laryngology and indelible marks on the entire social structure. The last name mentioned above stands out prominently as that of one who was a benefactor to the children of his generation and to those of future generations. He lived to see the benefits of his wonderful work. The name of Dr. Joseph O'Dwyer is known the world over for the invention of the O'Dwyer intubation tube and the instruments for its introduction and extraction, which were quite as wonderful as the tube itself. Whether intentionally, coincidentally or providentially, the perfected tube came at a time when the world stood aghast at the terrific and appalling mortality caused by laryngeal stenosis of diphtheritic origin.

Antitoxin was in its infancy. Although heralded the world over as a curative agent, it was looked upon with great suspicion, and no doubt justly so. It had its bitter enemies among the most famous men in the profession, including well known pediatricians. Their watchword was "wait." But it was very heartbreaking to be obliged to wait when the Grim Reaper was so devastatingly at work. To those of us who had seen the havoc wrought during the years when there was nothing of any avail to be done, there was great temptation to test the new remedy, hazardous though it may be. Improvement came rapidly and the laboratories were soon giving us an antitoxin that could be used with greater safety, yet was far from perfect. The immunizing dose must not exceed 500 units and the initial dose not more than 2000 units. Even this was attended with some danger, the reactionary effect frequently being a very alarming purpura hemorrhagica. When called to a case almost moribund with stertorous respiration and rapidly becoming cyanotic, to introduce the O'Dwyer tube was to see a modern miracle and a snatch from death; then the antitoxin. In a few hours the membrane would fairly melt away and the patient be well on the road to recovery. The intubation set is now laid away upon the shelf, thanks to the perfected development of antitoxin and its later forms; the dread disease is fairly conquered and will soon be extinct, but the name of our fellow founder, Dr. Joseph O'Dwyer, will ever be remembered and associated with others who have been benefactors to their race.

There is another surgical achievement which should be credited to this Society, performed at the Manhattan Eye,

Ear and Throat Hospital in the early days referred to, and which should not go unrecorded here. As far as my investigation has been pursued, it was the first case of complete laryngectomy to be performed in this country successfully, coupled with the placement of an artificial larynx with the production of a speaking voice. The case was one of sarcoma of the larynx with no apparent infiltration to any adjacent tissue. Of this we could not be sure, but it was an emergency that seemed permissible and the six months of comparative comfort the patient received warranted the operation; further than this, it was a demonstration that the operation was feasible. After six months it was apparent that the disease was extending to other tissues, although the original site appeared healthy. A more general operation was later performed by Dr. Robert Abbe, from which the patient did not recover. I would not again recommend the operation for a case of sarcoma. Such an operation in these days of surgical miracles does not appear unique, but to the pioneer it became a landmark.

This article may be considered as a sort of preview of Dr. Harris' paper, referred to at the beginning of this early history. I would, therefore, refer the reader to his account of the Society from the time when the 11 founders came together for their first consultation at the home of Dr. Robert C. Myles. Harmoniously of one mind, looking toward the coalescence of the trilogy, the combined interests smoothed the path and made the traveling a delight.

As I write this I am reminded of Edward Bellamy's "Looking Backward," viewing in retrospect the small beginnings up through the handicraft age to the machine era, and now when we think we have reached the top rung of the ladder, we look back and ask, "What next?" We do not need to go forward in imagination to the year 2000 to see marvels of medical and surgical science. We have them right now and here in the Triological, which has nobly contributed its share. But we have not yet arrived; there is as much to spur ambition now as in the beginning. I wonder if the things that are to be will be as satisfactory as the things that have been. Congratulations upon the past achievement and best wishes for the future.

1600 Steele Street.

FURTHER CLINICAL OBSERVATIONS ON THE INFLUENCE OF HYGROSCOPIC AGENTS IN CIGARETTES.

DR. FREDERICK B. FLINN, New York.

Flinn[1] in a report of a series of clinical observations on the irritant effects of cigarettes in which glycerine and diethylene glycol had been used as hygroscopic agents showed that the latter were less irritating. Subsequently, Wallace[2] said, "The results of the work are shown in the accompanying table and charts. From these it is seen that the agreement with those of Mulinos and Osborne is remarkably close. The correctness of their statement that the smoke from cigarettes which have been made with diethylene glycol as the hygroscopic agent is less irritating than those with glycerine is thus confirmed. Further, it may be stated that the duration of irritant effects from the cigarettes treated with glycerine is much longer than that from those treated with the diethylene glycol. With the experimental basis thus established, the clinical results obtained by Flinn are what would be anticipated and can be accepted as correct."

Recently voluntary reports have been received from two laryngologists who have been studying the effects of smoking on a number of their patients.

Approximately 100 patients were used as subjects. They were all smoking one of the popular brands of cigarettes, all of which are believed to contain glycerine as the hygroscopic agent. Careful case histories had been kept up to the inception of this study. The subjects were instructed to smoke only a brand of cigarettes known to use diethylene glycol as the hygroscopic agent. In order to obviate the effect of differences in tobacco, method of manufacture, etc., cigarettes of precisely the same composition but in which glycerine was used instead of diethylene glycol were obtained, and used by the subjects as controls.

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Although the previous reports gave strong indications of the results to be expected, yet it was most surprising in these later reports to see the conclusive nature of the effects of the hygroscopic agents used. Following are a few examples of the results:

Case 1: Mr. G. G. H. *Symptoms:* Gives a history of severe coughing spells and sore throats; smoking about 30 glycerine treated cigarettes daily.

Examination: Revealed a red, dry, glazed looking pharyngeal mucosa, some redness in the larynx as a whole, cords normal. I put patient on diethylene glycol treated cigarettes, and after a few days his cough disappeared and his throat and larynx were markedly improved. I later put patient on glycerine treated cigarettes and immediately his original symptoms returned.

Case 2: Mrs. E. V. *Symptoms:* Patient complains of an irritating cough and a sore throat. Patient smoking glycerine treated cigarettes.

Examination: Reveals marked redness and congestion of the pharyngeal mucosa. Larynx normal. I placed patient on diethylene glycol treated cigarettes and after a few days her symptoms disappeared, and on re-examination I noticed a decided improvement in the redness and congestion of the pharynx.

Case 3: Dr. T. C. B. *Symptoms:* Gives a history of a raw throat with paroxysms of coughing. He has always been an inveterate smoker of glycerine treated cigarettes.

Examination: His throat revealed a marked congestion and redness of the pharynx and uvula. I had the doctor smoke some diethylene glycol treated cigarettes, and the rawness and coughing soon disappeared. I experimented at different times with glycerine treated cigarettes, without his knowledge, and each time he used the glycerine treated cigarettes his cough and rawness returned.

Case 4: K. S. R. Irritating cough for several years. History of mild tuberculosis in 1918 and 1919—chest checked and X-rayed each year since, no return of active processes. Tonsils out July, 1932. December, 1935—cough more persistent, vitreous opacity in left eye and accentuated cough, strep-

tococcus hemoliticus in sputum—vaccine twice each week, almost continuously since January, 1925, which seems to improve cough. Known allergy to certain foods, but 120 intradermal tests made in the summer of 1935 were negative. From January to March, 1936, lost 10 pounds; developed acute bronchitis April 15, 1936. For two months cough very much worse, often sits up in chair for two or more hours coughing constantly at night. Streptococcus vaccine did not seem to improve cough. Smoking about 30 glycerine treated cigarettes each day. First two days on diethylene glycol treated cigarettes no cough at all. Cough greatly improved unless inadvertently encounters some allergic enemy in food.

To report on the findings in each case would be but a repetition of facts shown in these typical cases. Some subjects took longer than others to show the improvement, but the trend toward improvement was definite.

CONCLUSIONS.

The new studies reported to me by these doctors confirm my previous report. I have received similar reports from other doctors with a smaller number of observations; one of them said: "This study of glycerine treated cigarettes shows that they produce considerable irritation to the throat and in many cases are responsible for the paroxysms of coughing that many cigarette smokers complain of. After putting them on diethylene glycol treated cigarettes they had less throat irritation and the paroxysms of coughing promptly disappeared. In practically every case, except in those cases where there was definite pathology, my patients were markedly improved."

Laboratory[2,3,4] and clinical[1] studies are in agreement that cigarettes containing diethylene glycol are less irritating than those containing glycerine.

1. THE LARYNGOSCOPE, Vol. XLV, No. 2, 149-154, Feb., 1935.

2. *Arch. Otolaryngology*, Vol. 23, No. 3, 306-309, March, 1936.

3. *Proc. Soc. Exp. Biol. and Med.*, 32, 241-245, 1934.

4. *N. Y. State Jour. Med.*, Vol. 35, No. 11, 590.

Columbia University.

AMERICAN OTOLOGICAL SOCIETY.

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(Continued from December issue.)

The Nature of Bone Conduction As Shown in the Electrical Response of the Cochlea. Dr. Ernest Glen Wever and Dr. Charles W. Bray.

In these experiments the fundamental nature of bone conduction has been investigated by comparing the electrical response of the cochlea as produced by this form of stimulation with that produced by air-conducted sounds. This comparison is made, first of all, in terms of the functional relations between the magnitude of the cochlear response and the intensity of sounds conducted to the ear by the two methods. A further comparison is made in terms of the relative effects of lesions in the middle ear.

Electrical potentials from the round window of the guinea pig were observed by means of a cathode ray oscillograph on stimulation with sounds from a loud-speaker and from a bone-conduction receiver. For each type of stimulation the intensity was determined in absolute units.

With frequencies of 300, 1000 and 3000, the results indicate for each frequency a general similarity in the intensity functions for both air- and bone-conducted sounds. The curves have slopes which are the same, or nearly the same, for the two forms of stimulation. One feature of the air-conduction curves, that of bending over at high intensities of sound, is not shown by the bone-conduction curves, but the reason for this difference is uncertain; the intensity of stimulation by bone conduction was not sufficient for a thorough exploration of this portion of the response curves.

Further results reveal the effects of lesions in the middle ear on both air and bone conduction. After the incudostapedial joint of the ossicular chain was broken, the responses to bone-conducted sounds were changed much less than those to air-conducted sounds. In about half the cases, the responses for bone conduction showed no significant alteration; in the remaining cases, decreases up to 20 decibels were found. The responses for air conduction showed decreases which ranged from 50 to over 80 decibels, with most cases between 60 and 70 decibels.

Following the study of middle-ear lesions, the blood supply of the cochlea was eliminated and the animal was killed by pithing the medulla. Immediately after this operation the responses for both air and bone conduction showed a rapid decline.

The results show, in quantitative terms, the part played by the middle ear in the processes of air and bone conduction. Destruction of the ossicular chain causes a serious impairment of sensitive to air-conducted sounds, but has relatively little effect upon sensitivity to bone-conducted sounds. On the other hand, the two kinds of conduction give rise to essentially similar processes in the inner ear.

Dr. Heinrich Kobrak: The two muscles of the middle ear react under conditions to sound with tetanic contractions. The length of the contraction is determined by the duration of the tone, the strength is dependent upon the intensity and the frequency. The movements of both muscles can be easily observed in the animal (rabbit). In the human the contractions of the stapedius

muscle have been seen in a number of patients who have a perforation of the posterior half of the eardrum. Due to its lawful muscular movements this reflex offers a suitable means of testing cochlear function objectively, both in the animal and in the human.

Studying the role of the ossicular chain for bone conduction the following observations have been made:

Destruction of the drum membrane, hammer or incus does not diminish the cochlear function for bone-conducted sounds. On the contrary, a slight but definite improvement of bone conduction has been found.

After extirpation of the stapes an irritation of the cochlea by bone-conducted sounds can be still demonstrated. Bezold's conception that bone-conducted sounds only enter the cochlea via the stapes has, therefore, not been corroborated in the animal experiment.

In producing lesions of the sound conductive apparatus, a strong impairment of function for air-conducted sounds has been observed. Since there is no parallel loss of bone conduction the normal ratio between air and bone conduction is reversed (negative Rinne in the animal experiment). Lesions in the inner ear diminish cochlear function for air- and bone-conducted sounds alike (diminished positive Rinne in the animal experiment).

The conditions of the Schwabach test can be repeated in the animal by testing bone canal under normal conditions and after a lesion has been made. The observed increase of bone conduction in middle ear lesions is real, i. e., it is not produced by masking phenomena. The reflex shows no change in the presence of a masking tone, provided the masking tone itself is under the tensor threshold. Compared with the human as to hearing sensation, there is a definite masking effect if one uses the same acoustic stimulations as used in the animal experiment. In the human the stapedius reflex observed in the presence of a masking tone did not show any change, while the hearing sensation of the same person equally tested did give the phenomenon of masking. The acoustic irritation originating in the cochlea and determining the strength of the tensor contraction apparently is equal in the presence or the absence of a masking tone. Therefore, the phenomenon of masking is apparently not located in the peripheral sense organ.

The role of the intralabyrinthine fluid for sound conduction has been studied in the following experiments: If one drills a very fine hole in the apical part of the cochlea of the rabbit one can observe the escape of fluid and the functional changes connected with it. The lesion of the cochlear structures can be kept localized strictly to the place of the drill-hole, as far, at least, as is demonstrable histologically. A loss of function, increasing with the progressive escape of labyrinthine fluid, can be observed. The effect is about equal in bone and air conduction. If the drill-hole is allowed to heal, one can observe remaining function, since the inner ear apparently is now under more favorable hydrostatic conditions.

E. A. CULLER, Ph.D.: Our investigations in animal hearing at the University of Illinois have dealt primarily with other phases of audition, so that I have little to contribute on bone conduction. 1. Apparatus: We have used the two receivers, Western Electric and Acousticon. The efficiency or output of each was tested, at the seven octave frequencies from 125 to 8000 cycles, by attaching it to a silvered quartz-crystal bar and measuring the electric potentials therein generated by the mechanical impacts. The Western Electric receiver is fairly uniform in response from 250 to 4000 cycles, dropping off at 125 and 8000. The Acousticon is less uniform, being more efficient than the Western Electric from 500 to 2000, but less efficient at the extremes (125, 4000 and 8000). 2. Test Procedure: With one cochlea destroyed, the dog is first trained to respond to air-borne sound. A silvered screw is then turned directly into the bone of the zygomatic arch, and the bone-receiver attached. In this way firm and uniform contact is assured. To make sure that we are testing true auditory thresholds, and not merely tremor or tactile-sensitivity, the remaining

cochlea is destroyed after all desired tests are completed and the animal's response again measured. In one case, for example, the bone-limens with cochlea were —57, —75 and —51 decibels below our standard sound; whereas the bone-limens without cochlea were —19, —22 and 0. The cochlear thresholds are, therefore, far below the noncochlear; within that interval one may reasonably assume that the animal is responding to sound and not to tactile stimulation. 3. A few preliminary observations, not systematic nor definitive, are given in the full report.

DR. SIMON DWORKIN: As tested by our conditioning method, totally deaf cats perceive "sounds" when placed in an intense sound field. The frequency limits are from below 100 cycles to 1000 cycles, where a sharp cut-off occurs. The threshold intensities between 100 and 800 cycles vary from 105 to about 80 decibels above that of normal hearing at 1000 cycles. The responses cannot, therefore, be attributed to the pain or feeling levels established by Fletcher and co-workers. Most probably these intense sounds are perceived as follows: 1. At frequencies from 100 to at least 600, by tactile receptors in feet and legs. 2. At frequencies from 100 to 1000, by tactile receptors in the skin, the animal's fur has great ability to pick up and pass on these tremors.

An Experimental Study of Bone Conduction. Dr. Walter Hughson.

Changes in electrical potential in the auditory nerve of the cat, produced by bone conducted stimuli, have been investigated. The technical difficulties inherent in such a study, magnetic and electrical interference, mechanical vibration of electrodes, cross effect, etc., have been checked and overcome. In order to evaluate all phases of the problem it has been found necessary to use a nerve rather than a cochlear electrode. In an open bulla bone conduction is definitely reduced and this effect becomes more apparent when the septum is removed. To obtain constant conditions of stimulation a metal screw is put in the hard palate and the receiver tip placed on the head of the screw with a pressure of the receiver's own weight. One ear is destroyed after the control bone conduction audiograms for both ears have been made. Frequencies of 180, 250 and successive octaves to 8000 d.v. are used in all tests. Departure from the control level in decibels of intensity, whether improvement or impairment, is then measured for each experimental condition.

It was found that bone conduction was actually improved after the external canal had been blocked with vaseline gauze. This effect was manifested only if the bulla had been left intact. Air conduction was very much impaired. After removal of the gauze there was a prompt return to normal.

Incision of the tympanic membrane produced a marked effect upon bone conduction. With an extensive incision bone conduction was entirely lost for the two low frequencies, while air conduction was only impaired. In contrast to these findings, separation of the incudostapedial joint results in a complete loss of air conduction, while bone conduction shows only slight impairment.

In another series of experiments tension was exerted upon the tensor tympani tendon. Impairment in transmission is in direct proportion to the amount of tension used. Although both air conduction and bone conduction are affected, the latter response is impaired to a greater extent than the former. In addition, bone conduction fails to return to its control level when the tension is released.

This data, obtained from a large number of experiments, is presented more as a series of observed facts than as an effort to establish any theory of bone conduction. Many factors enter into the production of the phenomenon. The effective mass of the ossicular chain, its elasticity, phase changes in vibrations passing through the skull and ossicular chain, impedence of the tympanic membrane, must all be taken into consideration in attempting to make any final analysis. On the other hand, the results obtained indicate very definitely that the conductive mechanism of the ear in experimental animals plays a major part in transmitting bone conducted stimuli to the sensory cells of the

cochlea. So important is this particular route that transmission by bone conduction at a certain standard intensity of stimulus can be completely eliminated by experimental procedures carried out upon middle ear structures. The demonstration of this fact depends entirely upon elimination of the possible sources of error emphasized above. The fact that response by bone conduction can be so changed attests to the effectiveness of the precautions taken in these experiments.

End Organ Deafness in Dogs Due to the Application of Certain Agents to the Round Window. Dr. E. P. Fowler, Jr.

Changes both by the Wever and Bray method and by histological observation were reported in dogs whose round windows have been treated with NaCl and CaCl2. In addition, there was actual loss of hearing in dogs whose round windows were treated with NaCl. It was suggested that some of the cases of nerve deafness which occur in long continued suppurating ears, might be due to the poisoning of the hair cells in the lower turns of the cochlea by electrolytes seeping through the round window.

Dr. Moses H. Lurie: I am much pleased to find that in a number of ways Dr. Fowler has confirmed some of the work that we have reported in the past year. I was interested in his attempt to produce deafness by chemical means and to get the same histological result we have obtained by exposing the animals to loud tones. He also shows a similar condition that we are beginning to notice in some of the animals that we are breeding, which are congenitally deaf. I am sorry I have none of the slides of the congenitally deaf animals to show, but do have slides showing conditions very similar to what Dr. Fowler has demonstrated.

The loss of hearing was greatest with the greatest amount of destruction present. In the second turn of the guinea pig's cochlea, we had our greatest loss of from 40 to 60 decibels, while our least loss was towards the apex; and as the external hair cells began to come back into the picture so did the hearing of the animal begin to come back.

Dr. Fletcher: What was the sound?

Dr. Lurie: The sound was 2,500 for about a month at an intensity of 100 decibels.

The electroaudiograms on this animal showed a consistent loss, between 40 and 50 decibels, throughout the range up to 8,000.

Here we have two types of deafness, one due to a disease, the other one due to exposure to a loud tone for a considerable length of time, and now Dr. Fowler gives us the chemical results, something I have tried to do by feeding animals with chemicals, but I have not succeeded as yet in obtaining results.

(A number of slides were shown to illustrate the points made.)

Dr. E. L. Ross: A little over a year ago I reported some work on the use of chemicals in the middle ear and observed the effect on the vestibular function. The placing of cocain, novocain, quinin and salt into the middle ear, for a period of about one-half hour, produced decided changes in the vestibular function.

This past year I have employed the toxins of a number of different bacteria in the middle ear and have had some rather interesting results. The toxins used were from streptococci, staphylococci, pneumococci, tetanus, scarlet fever, and diphtheria.

This material was put into the middle ear of dogs for a period of one-half hour. These animals were observed for a period of a week or more. From the staphylococci, streptococci, pneumococci and tetanus no results were

obtained; that is, the effects were negative. The toxin from scarlet fever showed a reduced reaction of the vestibular apparatus. In the course of four days the diphtheria toxin produced death of the vestibular mechanism.

Because this destruction of the vestibular function was so absolute and we saw evidence of deafness in these animals, we ran a series of animals that were roughly conditioned for hearing, using the diphtheria toxin in the middle ear. In the course of four to five days, absolute deafness was produced by diphtheria toxin.

Report of the Board of Trustees of the Research Fund of the American Otological Society. Dr. John B. Rae.

I am quite certain that every member of the Society will share the deep regret of the Board of Trustees of the Research Fund that this report is made by substitute rather than by Dr. Arthur B. Duel, who was Chairman of the Board at the time of his death.

The vacancy in the Board, due to the regretted death of Dr. Edward B. Dench after long and faithful service, has already been filled by the election of Dr. John R. Page, a former President of the Society.

Following is an epitome of income, expenditures and allotments since the report of last year:

The present value of the Permanent Endowment Fund is approximately	$300,000
The estimated income for the year is	$ 14,500
Balance from last year is	600
Total available income for year approximately	$15,100

The Secretary's Report From the Board of Trustees of the Research Fund of the American Otological Society. Dr. J. Gordon Wilson.

Dr. Wilson referred to the conspicuous ability and enthusiasm which the various investigators had shown in the work. The studies progress along two main lines: Pathologic and Physiologic.

I. Pathologic studies have been concerned with the influence on the pathogenesis of otosclerosis, of developmental abnormalities, of infections in the fetus and in early infancy, and of nerve lesions. Investigation of developmental abnormalities follows as a subsequent stage to previous studies in embryology. As this subject is of fundamental importance it is specially referred to at the end of this report.

Dr. Blast continues his investigations on the petrous bone in the late fetal stages and in early infancy; also on the endolymphatic duct in man and in the guinea-pig to determine the angle between the utriculoendolymphatic duct and the common endolymphatic duct in order to determine the functional possibility of the utriculoendolymphatic valve. His studies in the "Fissula Post Fenestram" show that in 27 per cent it is present, but in about one-half of these it is a mere indication. With Dr. Eysler, professor physiology at Madison, he is studying the significance of the Wever and Bray phenomenon, referred to later.

Dr. Bast continues his investigations on the petrous bone in the late foetal between the cochlea and the stapes. He has studied the varieties of cartilage there present, the formation sclerotic bone and its relation to the fissula and to the blood vessels. A paper has been published on Paget's disease of the petrous temporal, a preliminary study to compare this disease in the petrous temporal with its manifestation in other parts of the skeleton. Since into the picture of otosclerosis there comes the stapes, and as no adequate description of its development and its abnormalities could be found in the literature,

Dr. Anson is studying, with the help of models, changes which occur in this bone from a 50-day embryo to the age of 70 years. There has been made under his supervision a large model of the petrous bone, showing the relation of the air cells to the Eustachian tube and the marrow spaces of the petrous tip.

Dr. Marvin Jones continues the work he was asked to do by the Committee, namely, to collect material for information regarding the pathogenesis of otosclerosis. In keeping with this Dr. Hemsath, working with Dr. Jones, has published an article on intrauterine and neonatal otitis media. In some there were found traces of amniotic fluid in the middle ear, showing a foreign body reaction without organisms; in others, evidence of pathological invasion of bacteria.

Dr. Fowler, Sr., and Dr. Fowler, Jr., continue their audiometric examinations and the study of microscopic sections from individuals on whom these tests were made. They discuss Dr. Albert Gray's hypothesis of the presence of definite degenerative changes in the VIIIth nerve in cases of otosclerosis, and cannot confirm Gray's findings. They have made an important observation on the origin of acoustic neuromas, namely, that such tumors, large and small, seem to arise from the base of the modiolus and suggest a traumatic origin.

II. Physiology. One feature common to all reports is the use of drugs to determine the function of various parts of the peripheral auditory apparatus. Since another member of the Otological Society is carrying on and has already published results of the application of various drugs to the vestibular mechanism, it is gratifying to find the double mechanism of the ear, following the application of stimulants and depressants, being studied by members of our Society.

Dr. Hallowell Davis and his co-workers at Harvard, who have published several papers, say: "We feel that the outlines of the theory of hearing are now on a firm, experimental basis. Pitch perception is determined by the stimulation of particular nerve fibres and the degree of loudness is given by the total number of active fibres. We have begun experiments to endeavor to detect histological degeneration of physiological depression of the organ of Corti produced by quinine and certain other drugs. Also we are collaborating with the Department of Neuroanatomy in a study of the congenital abnormalities of the auditory and vestibular apparatus in waltzing guinea-pigs. We test electrically the functional state of the auditory apparatus and then examine the cochlear and labyrinthine end-organs histologically, while our anatomical colleagues study the corresponding neural tracts. The genetic aspect of this problem promises to be one of great general interest."

He points out that masking does not occur in the cochlear response. Masking is neural, the cochlear response is not neural, but is generated by the sensory cells of the organ of Corti, and not by the nerve cells or nerve fibres.

Dr. Bast has studied the effect of various drugs placed in the middle ear (round window or oval window region) on the transmission of the Wever and Bray phenomenon, to determine whether the phenomenon has its origin in the nerve end-organs or not. "Starting with the supposition that such drugs as cocain, etc., will deaden the nerves and end-organs; then if the phenomenon originates in the organ of Corti or if the phenomenon is an index of hearing; it should be obliterated by these drugs. Our results show that the phenomenon is not obliterated when these drugs are applied. All these drugs depress the low frequencies and augment the high frequencies. This cannot be explained on the basis of nerve involvement. These drugs may produce a change in the permeability of some membrane or may produce pressure changes across some membrane, resulting in lowered potentials for low tones and raised potentials for high tones."

From McGill Dr. Dworkin reports on the measurement of hearing loss in cats after middle ear lesions and after damage to the apex of the cochlea

He further reports a neurosis developing in dogs when they were exposed to min.mum audibility stimuli.

A summary of the anatomical work of Bast at Madison, and of Anson and Wilson at the Northwestern University Medical School. The anatomical facts are determined; for the defects of the hypothesis Dr. Wilson is responsible.

The bony periotic capsule develops in the usual manner from primitive connective tissue, first into cartilage and later into bone. The dense connective tissue perichondrium develops periosteal bone similar to that found in the long bones of the skeleton. The cartilage is displaced by primary endochoral bone, in which is embedded intrachondrial bone (cartilage islands). This primary bone is characterized by its web-like strands, with the bone cells laid irregularly but in a direction more or less longitudinal. Elsewhere in the skelton, e. g., are long bones, this primary bone is replaced by lamellar bone around Haversian canals. But not so in the petrous capsule, here Haversian lamellae are few in number. In short, the fully formed periotic capsule consists of primary endochondral bone, reticular and not lamellar, an osseous tissue more resistant to pressure than cartilage and more pliable than lamellar bone.

In the fetus in the area between the stapes and cochlea there is a communication between the vestibule and middle ear filled with loose connective tissue and surrounded by cartilage, called the fissula antefenestram. This fissula usually disappears and the anatomical structure reaches a formation similar to that elsewhere in the periotic capsule. But the fissula may not disappear; the normal progress may be interrupted and may show developmental irregularities spreading into the surrounding spaces, for instance, the loose connective tissue may persist from fetus to late adult life; the cartilage, normal in the fetus, may increase and persist during life, irregular in size and location; large marrow spaces showing invasion by cartilage may be seen; there may be present abnormal bone, irregular in outline, varying in amount and sharply separated from the tissue in which it lies. In short, in the region between the stapes and cochlea there may be present, at all ages, tissue of various histological types, including sclerotic bone, irregular in outline and of varying dimensions. Though the physiological significance of the fissula is not known, the outcome of this diversity of the tissue around it suggests that in certain cases the tissue in this area remains in a state of instability, insofar as it does not reach the condition normal to the periotic capsule. The primitive constituent cells may remain unchanged in type, or after a varying progress further development may be arrested, or this progress may be intermittently resumed. In the resulting picture there is seen a structure with irregular anatomical dimensions of varying histological pattern.

Morphologists believe that instability of tissue is of importance in the genesis of abnormal growths. Unstable tissue is known to be influenced by environmental factors, and further, that a stimulus which intermittently calls forth the reactivity of an unstable tissue need not be a particular stimulus. That the tissue in this fissular area retains potential activity of growth is evidenced, apart from clinical evidence, by the ability of cartilage to spread into adjacent marrow spaces, and by the formation of sclerotic bone of abnormal type so sharply marked off from the surrounding bone or tissue as to appear independent of it.

Our hypothesis is that this instability of tissue is the fundamental basis of otosclerosis and that environmental stimuli, using the word environment in its widest sense, disturb the instability from time to time and lead to a growth abnormal for that locality. This hypothesis is in agreement with modern scientific opinion and fits in with our clinical knowledge of otosclerosis. Anatomically unstable tissue may be present, and intermittent activity is recognized by clinicians. There is evidence that the vascular factor may play an important part. The principal vascular supply of this area, both in fetus and adult, is from the tympanic cavity. Additional support for this hypothesis is given by a study of Paget's disease of the petrous temporal which, as elsewhere in the body, shows destruction of bone, followed by reconstruction by

a different type of bone. While the compact cochlear capsule may be attacked, it seems to present resistance to the progress of the destructive forces, except in the area between the stapes and cochlea. Our argument is that in and around the fissula antefenestram, between the stapes and cochlea there may persist into adult life, tissue representing stages in the growth of the normal capsular bone. That, morphologically, this tissue is unstable and under the influence of stimuli may result in irregular growth. In short, morphologically, unstable; clinically, vulnerable.

A Method for the Early Detection of Otosclerosis — A Study of Sounds Well Above Threshold. Dr. Edmund Prince Fowler.

The idea which inspired these experiments was that the footplate of the stapes would not be impeded at its normal threshold excursions, even though there was a partial involvement of the annular ligament, but that for greater movements as for greater excursions (those occasioned by louder sounds) the stapes would be impeded.

Threshold hearing may be normal, but sounds above the critical intensity would be sensed below the normal expectancy. Tests were devised to detect the critical amplitude of this impedance. The background was laid upon this hypothesis and several clinical observations regarding the inadequacies of minimum audibility as a criterion of hearing acuity for loud sounds. Several peculiarities of the hearing for the louder sounds are mentioned, and the lesions which were suggested by such. Also the discrepancies in masking and bone conduction data, some of which may be explained by the author's experiments.

Some 18 or 20 ways were devised to measure the critical amplitude where the "hobble effect" occurs. Most proved unsatisfactory, especially with inexperienced observers, although in several instances the "hobble effect" was found in members of families with histories of otosclerosis.

A method of alternate binaural loudness balance was devised, which not only would detect the "hobble effect," but which brought to light the fact that in certain people a poorer ear may hear loud sounds almost or quite as well as the normal or better ear, in spite of the fact that the threshold of hearing for the two ears may be separated by even as much as 60 decibels of loudness. Both air and bone conduction and combined air and bone conduction were used to bring out the data.

The experiments produced four distinct phenomena which were reduced to quantitative measurements and shown in graphs according to lesions.

1. In certain people a constant difference (or no difference) in the loudness is necessary for binaural balance at all intensities.

2. In certain people a changing difference in the loudness is necessary for binaural balance at different intensities, i. e., a poorer ear which appears to hear loud sounds disproportionately well as compared to faint sounds. (There is a greater increment of loudness with similar changes in intensity above threshold than in the better ear.)

3. A combination effect of No. 1 and No. 2.

4. Anomalies in intensity discrimination, i. e., in certain ears at critical intensities, small loudness changes appear greater or less than average normal, or greater or less than to the opposite ear.

The graphs suggest several applications and possibilities aside from those in mind, particularly as to animal experimentation and differential diagnosis of intracranial lesions, and lesions in the different parts of the auditory nerve, cortex, ganglia, and end-organs. The methods have proven useful in overcoming uncertain observations caused by inattention, nervousness, tinnitus, etc., because sounds 15 or 20 decibels above threshold are so loud that they are

very definitely heard, easily balanced binaurally, and are not masked or confused by the tinnitus.

Genetics in Otosclerosis. Dr. Max A. Goldstein.

The detailed investigation of this extensive otosclerotic family, with many ramifications, serves a six-fold purpose:

1. Evidence that otosclerosis is an hereditary disease of the ear and is transmissible, generation after generation, without a break.

2. That otosclerosis may develop at a very early age and that its insidious onset may not be easily diagnosed.

3. That otosclerosis, of marked degree, in its usual onset, may simulate congenital deafness.

4. That there is no justification by evidence produced in a great number of cases that congenital deafness is an hereditary disease.

5. That there is justification for the consideration of otosclerosis as an hereditary disease.

6. That there is a possibility that otosclerosis may be developed in embryo, and that such embryonic otosclerotic pathology may be confused with, and show aspects of similarity to congenital deafness.

These observations are the result of this extensive survey of a detailed familial history and may offer data for the further development of a field which may throw some light on the origin of both congenital deafness and otosclerosis.

Some 12 years ago Gustave Alexander reported the case of a girl, age 10 years, in whom he had made an antemortem examination and a clinical diagnosis of otosclerosis. This girl died of some acute condition, and the temporal bone in the laboratory confirmed the clinical diagnosis, with its picture of typical otosclerosis as we understand its pathology at present. Dr. Stacy Guild, in his routine work of sectioning temporal bones at Johns Hopkins' Otological Laboratory, reported the case of a child, age 1 year and 10 days, in which he had diagnosed under the microscope the findings that we identify as otosclerotis in the labyrinth capsule.

The following conclusions can be made:

1. Deafness occurs in all four generations, an unusual condition in hereditary transmission. No previous genetic studies of otosclerosis have been traced back more than three generations.

2. When defective hearing occurs on both sides of the family there is: a. An increase in the per cent of cases; b. defective hearing at a lower age level.

3. There seems to be a relationship between other physical ailments, such as susceptibility to colds, sinus infection, nasal catarrh, poor teeth and progressive deafness. What this relationship may be has not been determined.

Discussion of Dr. Fowler's Paper. Dr. T. Gordon Wilson.

Dr. Fowler is justly regarded as an expert in the interpretation of audiometric graphs. His communications always repay careful study and the paper today is no exception. It is a preliminary report and is to be so regarded. It is a plea for measurement of hearing acuity by air and bone conduction, not only at threshold intensities, but at higher intensities, pointing out that such are capable of far reaching interpretation. The importance of early diagnosis in otosclerosis and the success which may follow its recognition is becoming more generally recognized; undue pessimism has been a great drawback

to success in treatment of this malady. It is known that the disease is subject to periods of quiescence; it is known that there are constitutional causes or environmental agents which arouse its latency or stimulate its progress; such facts suggest the possibility of control of the lesion. With early diagnosis before the pathological change is unalterably fixed, with recognition of the disturbing causes, we shall have advanced to a more hopeful frame of mind regarding a prognosis.

The underlying hypothesis enunciated by Dr. Fowler is, that whatever hampers the free movement of the stapes interferes with the conduction of air borne sounds. Where the fixation is incomplete very small movements may not be appreciably affected, neither would minimum audibility; but with greater movement of the stapes, that is, greater amplitudes, there would be an earlier noticeable defect in perception. To the former one may agree, the latter is arguable. That lesions other than otosclerosis lead to deficient free movement of the stapes we need not now consider.

Can we say definitely that the deficiency of hearing in otosclerosis is due in all cases to interference with free movement of the stapes? Are we to disregard cases in which there are all the clinical signs and symptoms of otosclerosis, including deficiency of hearing, and yet, pathologically, no analysis is present? Dr. Gray quotes such cases. On the other hand, numerous cases are in the literature with extensive areas of otosclerosis around the oval window, but with the annular ligament not affected and normal hearing. At present, the evidence available would indicate that without some stapes fixation deficiency of hearing does not occur.

Dr. Fowler's main contention is that in otosclerosis it is from the testing of the hearing at amplitudes greater than the minimum, where there is a larger movement of the stapes, that one will detect early deficiencies, and not from testing minimum audibility; to limit tests for early interference to threshold audibility is futile, for the hobbling of the stapes will have progressed some distance before this minimum is affected. If so, then the threshold of hearing (e. g., audiometric charts) no longer appears to be a dependable criterion to estimate freedom from disease. The hobbling effect is undoubtedly much smaller at threshold audibility but so also is the energy transmitted. Since in the stapes ligament, where the ankylosis occurred, we are dealing with a living tissue, will the hobbling effect follow ordinary physical laws? Such are a few of the disturbing questions which arise in one's mind. Dr. Fowler seems to anticipate trouble and wisely says this is fundamentally a question for the physicists and so as clinicians we happily may leave the solution of our doubts to them.

In support of this hypothesis the graphs which Dr. Fowler shows and which were made in cases of nerve and conduction deafness must be carefully studied before one can decide on the clinical importance of this interesting and valuable communication. It may well be that Dr. Fowler is pointing to a new road to an early diagnosis of otosclerosis. What I have said represents the questions which have arisen in my mind when reading the paper.

R. L. Wegel, Ph.D.: In his exploration of comparison of perception of intensity by two ears, Dr. Fowler has opened concretely a new field, which bids fair to give useful diagnostic information concerning differentiation between conduction and nerve lesions. His tests are convincing because they deal not only with threshold intensities, but also with the higher and more useful sound levels and because specific, easily measured results are obtained. Dr. Fowler has described the correlation of his tests with threshold acuity, and has also observed that abnormality of intensity discrimination is also related to his tests. In regard to the last point, it may be pertinent to examine the ability to discriminate intensity changes in low near the threshold, rises along a sigmoid curve to a maximum at moderate levels and remains constant as the intensity is further increased. In some cases, however, only the upper part of the curve is obtained. The absence of the lower part cannot be due to a conduction defect. It must be related either to a degeneration of nervous tissue

or to an abnormal distribution of it in the first place. Some curves also show anomalous behavior at moderate intensities. These may be associated with Dr. Fowler's "hobble," or with an odd distribution of nervous tissue.

JOHN C. STEINBERG, PH.D.: Although it has often been said that deafened people hear better in a noisy place, or hear loud sounds normally, Dr. Fowler, by comparing the sounds heard in normal and deafened ears, has obtained quantitative data on the effect. We have observed both the "constant" and "variable" type hearing loss in tests on people having one normal and one impaired ear. The variable type loss has been observed also in cases where both ears are alike and impaired over part of the frequency range. In this case tones in the normal frequency range are compared with tones in the impaired range. Many of the characteristics of the variable type loss are obtained when tones heard in an ear masked by noise are compared with the tones heard in the unmasked ear.

DR. I. H. JONES: Right after the war it seemed to me the desirable thing was to know how well the patient heard, and so we used the audion bulbs, as we then called them, as an amplifier.

In brief, from patients that I knew intimately, I was able to find that those with definite perceptive defects disliked amplifications; that those with purely conductive defects, or almost purely defective defects, liked it very much.

Then the question arose of measuring the degree by which they could hear well, the degree of amplification at which they could hear well; in this connection Dr. Fowler has mentioned the stapes motion. It does seem that the point is very well taken, because one-billionth of an inch, I am told, is sufficient amplitude at 1000 cycles for a drum membrane and the ossicular chain to move, in order to permit us to receive an audible sensation. On the other hand, my voice would be one-millionth, which is 1000 times greater. Consequently, it would seem very easily appreciated that a very faint, one-billionth of an inch, might get by, whereas one-millionth or greater amplification could not get by.

DR. STACY R. GUILD: Disregarding any conclusions that Dr. Fowler drew, tentative or otherwise, I wish to say that I think this is an important contribution to our methods for the study of hearing. He is obtaining by this new technique certain facts concerning balance between the ears, which however, we wish to interpret later. I wish to draw a clear distinction between tentative conclusions now and possible usage of the data obtained.

HARVEY FLETCHER, Ph.D.: I have been interested in experiments on loudness for the last 15 or 20 years. We have been confused in our thinking for a long time, but are gradually getting to understand what they mean.

I am very glad to see that the otological profession, particularly Dr. Fowler, is beginning to use loudness tests in making diagnosis. It seems to me this is one of the most important contributions to the field of functional tests that we have had in recent years. I think that, not only will it do some of the things that Dr. Fowler is expecting of it, but it might very well contribute some information which will give us a more clear picture of the mechanism of hearing, particularly on the nerve side of that mechanism.

Some years ago I made a recommendation which seems to have been taken too seriously. In Dr. Wilson's discussion, he questioned the practice of using 500, 1000 and 2000 cycles for getting the average hearing loss. I think I was guilty of introducing that notion, and it was done very innocently, and not with very much experimental background. It was done to give an answer to the question which is always asked by a patient: "What is my hearing loss?" He means by that, what is his hearing loss for speech.

We made some experiments on determining just what the hearing loss for speech was, in patients having various degrees of hearing loss. Then we made audiograms of about eight patients. It turned out that if you used 500, 1000 and 2000 cycles, and averaged the results for these frequencies, you obtained

very nearly the same value as if you actually made the speech tests. It probably will not hold for peculiar types of deafness. It was an approximate rule and I hope the otological profession will not take it too seriously.

DR. MOSES H. LURIE: I would like to congratulate Dr. Fowler on applying a method that has been used by Dr. Stevens, in the Department of Psychology in Harvard, on the question of loudness and the balancing method for discrimination of loudness in psychological tests.

It was on account of these tests (loudness tests) that we began to look for the question of difference in function between internal and external hair cells of the organ of Corti. From the degenerations that we have found in animals we began to believe that the discrimination of loudness and pitch depended more on the number of nerve fibres and the activation of the internal hair cells, than upon the external hair cells. The work of Dr. Fowler confirms this, that in the human the more intense the sound becomes, the better the discrimination of pitch.

DR. SIMON DWORKIN: Although a nonmember, I would like to add a comment or two on Dr. Fowler's paper. If I understood his general thesis correctly, much is based on the fact that the stapes at hearing threshold will have to move a very small distance, as compared with the movement at the higher sound intensities, and that the stapes, even when slightly impeded, might still be movable by threshold sounds, but be greatly hindered in responding to loud sounds. That seems logical enough when one thinks in terms of the bone, but if one thinks in terms of the stimulus necessary to set it in motion, I wonder whether the hypothesis would hold.

It has been shown, for instance, by the Bell Telephone engineers that at threshold, particularly for notes around 1000 vibrations, the energy required to move the stapes is exceedingly small, something of the order of 10 to the —6 watts. It is very hard to see that it would be possible, with that amount of energy, to move a bone that was in any way hampered, and therefore the threshold ought to be noticeably impaired as long as there is any fixation at all of the stapes.

DR. EDMUND P. FOWLER: Let me thank particularly Dr. Wegel and Dr. Steinberg in aiding me in this work. I am not a physicist, although I use the language sometimes glibly. It is by their help that I do it.

We know otosclerosis occurs very frequently without any deafness. It has been then clinically not possible to diagnose it. I believe the tests I have described may furnish aids to make it possible. Whether we have nerve deafness or not, the "hobble" hypothesis would still obtain, as well as if the nerve deafness was due to one thing or another, or whether it was due to otosclerotic pressure or pressure from other lesions.

Near minimum audibility, it is difficult to sense slight changes of sensitivity; but if we see these cases early this so-called hobble effect will not be found at near minimum audibility; it will be found some way above it. This was the thought: The possibility of detecting the impedance of the "hobble" not just before deafness is appreciable, but also some time before.

Fibrous adhesions around the stapes will cause, in one sense, a hobble effect, but this is not what I am referring to, because adhesions do not change the ratio of the increment of loudness. It remains relatively the same throughout the intensity scale. It does not suddenly change at a critical intensity.

With regard to bone conduction, I have also balanced bone conduction against air conduction; and in the cases we have observed, these also went up in parallel ways. When bone conduction is normal you have a normal reference to compare with the low reference of the deafened ear.

Dr. Steinberg showed graphs describing the same types of phenomenon that I showed to you. To me, as a clinic, it is difficult to grasp his graphs as well

as I can my own simple ones. I believe my diagonal lines show the balance difference better than the other methods of plotting, at least to clinicians.

As I understand it, Dr. Lurie, Dr. Stevens used only relative degrees of loudness, such as half-loudness; not equal balance.

DR. LURIE: He balanced the degree of loudness from one ear to the other in order to get discrimination of loudness and also discrimination of different characteristics of sound.

DR. FOWLER: Was it equal loudness?

DR. LURIE: It was equal loudness.

DR. FOWLER: I am glad to hear this.

Report of New York Committee on Otitic Meningitis. Dr. James G. Dwyer.

This is the second official report of the local committee in New York.

By far, the biggest problem we have is meningitis. The deaf person accustoms himself to conditions. We bury 97 per cent of meningitis. We are trying in a poor, feeble way to get ahead on the problem and I was just thinking, as I was sitting here, if one-tenth of the effort of some of us all over the country was devoted to a problem where it was a matter of life and death, we might get somewhere.

Since the last report in New York, we have had 22 cases with one recovery. We have been meeting regularly, three or four times, analyzing the different cases.

I am very proud to say that in the last year and one-half I think we have saved four lives by one of the points that we have been stressing, namely, that a cloudy fluid without organisms means one should operate and find the focus. We have saved four of them. I do not think there is any doubt about that.

The work at present is purely educational. We are not attempting anything in the way of treatment because we are limited, but we do believe that we can get tremendous help in early diagnosis of meningitis by the recognition of three simple factors: the blood count, the chemistry of the spinal fluid, and a recognition of the pathways of infection. When we have lost a case of mastoiditis we have lain awake nights, thinking of some error in our technique. From our postmortem work and from other observations and analysis of the histories, I have come to the conclusion that most deaths in mastoiditis are practically from the nose and throat; I say most. I believe the number of deaths due to injury of the dura or exposure of the dura is very small. We have had several autopsy specimens in which we definitely proved that the pathway was from the sphenoid sinus. I have over six in which we got partial autopsies, where we were able to take out the base of the mastoid and prove there that what we were essentially dealing with was an osteomyelitic process and not mastoiditis. Those cases were lost from the start.

A persistently high blood count in an adult, say, 30,000 to 35,000, in a mastoid or ear case, in which the chest is excluded, is going to end in meningitis, and you may find out from this blood count four or five days or a week in advance and be prepared. I have proved that time and time again.

Third, the question of cloudy fluid. We want to emphasize the point that a persistently cloudy fluid, loaded with cells but no organisms, is still a case that you can save. If you wait too long, meningitis will develop. As I say, I believe we have saved four of those.

I think the time has come when we ought to get busy all over the country and do the same work on meningitis that we are doing on otosclerosis. We have not any money in New York. We are doing it voluntarily, bearing the expenses ourselves. We go out, day or night, to any hospital that will call us.

Although I am reporting 22 cases on which we received histories, I know of at least between 30 and 40 in other hospitals who died, on whom we did not get reports. We are gradually trying to educate them.

We go out day or night to private or clinic cases; we try to establish the diagnosis and to get autopsy material. I think the time has come when the Society, as a whole, should establish a central committee whereby, as Dr. Beck is doing in the pathology section, we can get this pathological material assembled, have histories assembled and analyzed, and get the correct diagnosis in papers that are published.

At present we, as scientists, certainly ought to know when we are dealing with true meningitis and false meningitis, and we ought to make the diagnosis. I certainly believe it can be made 100 per cent from the spinal fluid. We certainly ought to talk all over the country in one common language of what is meningitis and what is not.

Dr. Samuel J. Kopetzky: Progress in handling meningitic cases in our hands has come from the study of the chemistry of the fluid and from the isolation (this is a preliminary report and is to be taken with all reservations) by our bacteriologist, of a substance having the nature of an enzyme. This we found in the cerebrospinal fluid of streptococcically infected fluid. It is a fibrolytic substance which creates the fibrinous deposits. Our efforts, of course, are being directed toward making a substance to defibrinize and keep the circulation of the fluid going. That is one step in progress which I want to report. A preliminary report on this was made to the American Society of Bacteriologists at its twenty-seventh annual session recently.

Reporting on the chemistry of the cerebrospinal fluid, of all the various kinds of meningitides I made comparisons with the normals and I show you the drop in the chlorides and the bicarbonate; carbon dioxide in the fluid is very marked in all types of meningitis.

The finding of the chemical quantity of these reactions in the fluid is not sufficient; there must be, at the same time, a parallel examination made of the same substance in the blood stream; the normal in the blood, let us say, of lactic acid as compared with the meningitic blood; in the cerebrospinal fluid, the normal as compared with the finding in the meningitic case; the normal sodium chloride, the drop in the meningitic; the normal carbon dioxide in the blood with the normal in the fluid, and the drop in the meningitis fluid; the small fall in the Ph as compared with the condition of the blood.

We have been able to keep meningitic cases alive for a time much longer than usual by the use of transfusions, very small transfusions, 75 to 100 cc. irrespective of the size and weight of the patient, because we found that this restores (we never knew why) the chemistry of the cerebrospinal fluid to furnish an environment in which the organism grows, less favorable to the invading organisms, which is in fact to a certain degree inhibitory to its development. After transfusion the lactic acid drops and the chlorides go up. This is a trend toward normal.

I have published a report in which the same factors shown on the previous charts are placed in juxtaposition to the clinical history and the temperature curve, and it was found that long before the clinical history clears up, the falling of the ratio toward the normal, as far as lactic acid is concerned, and the rise toward the normal, as far as the other factors are discerned, gives an indication of prognosis.

In the taking of the fluid, certain precautions should be remembered. The fluid changes very, very rapidly and, therefore, it is necessary to cover it at once with paraffin, at the bedside, so that there be no leakage or evaporation. Immediately start your examination for your tests. Take a second specimen for the study of the bacteria.

I think if in all the centres this work were undertaken, we might learn something more about streptococcic meningitis which might lead us toward

finding a curative therapy. There are more cures than are reported in the New York Committee, but they have not yet been turned over to Dr. Dwyer because of the necessary clerical help to copy the histories.

Audiometry and the Prescribing of Hearing Aids. Dr. Isaac H. Jones.

Audiometry has emerged from laboratory; now assumes its place as a clinical art. Contrast of eye and ear; optics long established. The problem with the ear was to determine the condition of all those parts we cannot see. The first advance came, not in auditory, but in vestibular studies; we measure objective responses of hidden structures. Optics impossible with precision instruments. Acoustics similarly needed precision instruments. Thirteen years ago the writers suggested ideal requirements, constructed 25 instruments, and now present a new audiometer, designed to meet the original requirements. Technique of examination. The details of prescribing a hearing aid. Improvements in hearing aids. Further studies in selective amplification.

Audiometry cannot reveal pathology, but can give data for diagnosis and the prescribing of the best possible hearing aid. We have long been able to secure a precise knowledge of the function of the eye. The turning and caloric tests and precise audiometric measurements at least give us the key to a similar mastery of the ear.

Dr. Horace Newhart: After what has gone before in the business meeting and because of the large number of physicists and acoustic engineers present, I feel that we can best serve this group by asking the President to call upon a guest, Dr. Henry E. Hartig, of the department of electrical engineering of the University of Minnesota, who, during the past two years, has directed extensive research in a field definitely related to the matter covered by this paper.

Dr. Henry E. Hartig: I have to report some data we obtained at the University of Minnesota in an investigation begun about two years ago. Dr. Newhart asked the cooperation of the department of electrical engineering at the University of Minnesota to make an examination of the various commercial carbon microphone hearing aids which were on the market. We proceeded to do that, first, in a preliminary investigation in which it became very evident that many of these commercial hearing aids could be eliminated immediately as having insufficient amplification, in some cases having actually a loss instead of a gain and, also, having very poor frequency response characteristics.

We finally narrowed the investigation down to four of the leading hearing aids of the portable type on the market, consisting of a microphone and an amplifier, and earpieces consisting either of the large watchcase type, or the ear fitting type, and others having the bone conduction type of receiver.

An ideal that has been expressed a great number of times in the past is that the hearing aid should have uniform amplification over a wide frequency range. Recently, as Dr. Jones pointed out, another ideal has been expressed which is that the hearing aid should have selective amplification; that is, amplification which varies with frequency in some way, corresponding to the patient's hearing loss.

Of course, it is obvious that unless hearing aids are available which satisfy either one or the other of these ideals, or some other criterion which may be set up in the future, the program is stopped right there. So, it is very important to investigate the characteristics of the hearing aids which can be obtained at the present time.

In investigating carbon microphones and in investigating acoustic problems in general, a great many difficulties exist, as those who have ever tried a thing of that kind very well know. I just want to impress on you that the investigation here reported on was carried on carefully. We had a great number of checks on our results and we feel that the results we here present are accurate and trustworthy.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

The annual meeting of the Council of the American Laryngological Association was held at the home of Dr. James A. Babbitt, in Haverford, Pa., on Jan. 9, 1937. The following members were present. Dr. Chamberlain, Dr. Wherry, Dr. Babbitt, Dr. Imperatori, Dr. Coates, Dr. Greene, Dr. Newhart and Dr. Shurly.

It was decided to hold the fifty-ninth annual meeting of the American Laryngological Association at the Marlborough-Blenheim Hotel, Atlantic City, N. J., on Monday, Tuesday and Wednesday, May 31, June 1 and 2. Dr. R. H. Stetson, Professor of Psychology at Oberlin College, will be the guest speaker.

This meeting follows that of the American Otological Society at Long Beach, N. Y., and precedes the meetings of the American Laryngological, Rhinological and Otological Society and the American Medical Association at Atlantic City.

The Association has lost by death the following members: Dr. John S. Fraser, Edinburgh, Scotland, Corresponding Fellow, May 11, 1936; Dr. Henry L. Wagner, San Francisco, Cal., Emeritus Fellow, Dec. 27, 1936; Dr. Robert C. Myles, New York City, N. Y., Emeritus Fellow, Jan. 1, 1937.

The Council recommended for election to Emeritus Fellowship, Dr. Harry C. Barnes, of Boston; for election to Honorary Fellowship, Dr. F. R. Nager, of Zurich, Switzerland; and to Corresponding Fellowship, Dr. Georges Portmann, of Bordeaux, France.

According to the Constitution there are but four vacancies to be filled and the Council has recommended for election to Active Fellowship, the following: Dr. Robert E. Buckley, 50 East 63rd street, New York City; Dr. Samuel James Crowe, 4332 North Charles street, Baltimore, Md.; Dr. Carlton Stewart Nash, Medical Arts building, Rochester, N. Y.; Dr. George L. Tobey, Jr., 270 Commonwealth avenue, Boston, Mass.

The resignation of Dr. Frederick E. Hasty, of Nashville, Tenn., from Associate Fellowship, on account of ill health, was accepted with deep regret.

Dr. Charles J. Imperatori was reappointed Abstract Editor.